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## **BASELINE RISK ASSESSMENT**

### **HOOKSTON STATION SITE PLEASANT HILL, CALIFORNIA**

**November 2004**

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\*from CTEH, 2004

## **1.0 INTRODUCTION**

As directed by the San Francisco Bay Region California Regional Water Quality Control Board (SFRWQCB), this report presents a baseline risk assessment (baseline RA) of chemicals detected on- and off-site near the Hookston Station Site in Pleasant Hill, California. In April 2004, a risk assessment was prepared and submitted to the SFRWQCB using SFRWQCB risk assessment methods (CTEH, 2004). In the April 2004 RA, concentrations of chemicals detected in indoor air, soil, soil vapor, ground water in on-site locations and chemicals detected in indoor air, ground water, and soil vapor in off-site locations were compared to SFRWQCB Environmental Screening Levels (ESLs). As defined by the SFRWQCB, ESLs are conservative levels of chemicals in environmental media that can be assumed to not pose a significant, long-term (chronic) threat to human health and the environment. Chemicals detected in on-site indoor air, soil, soil vapor, and ground water exceeded ESLs. In addition, chemicals detected in off-site indoor air, ground water, and soil vapor exceeded ESLs. The presence of a chemical at a concentration above its ESL does not necessarily indicate that adverse effects to human health or the environment may occur; instead, exceedance of ESLs suggests that additional evaluation is warranted.

This baseline RA is submitted to comply with new Task 8.b. Baseline Risk Assessment ordered by the SFRWQCB on August 12, 2004. The order states that

The Baseline Human Health Risk assessment, at a minimum, will quantitatively evaluate the cumulative risk to human health posed by exposure to contaminants derived from the subject site in air, soil, and ground water in both on-site and off-site areas.

A baseline RA differs primarily from the April 2004 risk assessment in that estimates of human exposure to chemicals of potential concern in each environmental medium are estimated and theoretical estimates of noncancer and lifetime cancer risk are calculated. The baseline RA presents estimates of exposure to on-site individuals in indoor air and soil and off-site individuals exposed to indoor and outdoor air and ground water.

As ordered by the SFRWQCB, the baseline RA evaluates risks posed by “contaminants derived from the subject site.” Because the Hookston Station site is not the sole source of chemicals detected in indoor air or ground water, analysis is presented in the baseline RA which allocates risk to only potential Hookston Station sources. The source allocation analysis was performed by ERM. The source allocation analysis of tetrachloroethylene and its degradation products in ground water is attached to the baseline RA as Appendix A. In addition, the risk posed by

tetrachloroethylene, trichloroethylene, and their breakdown products, regardless of source, is presented for the purpose of comparison.

Selected figures from the August 2004 Remedial Investigation report (RI) prepared by ERM (ERM, 2004) are attached to the baseline RA. These figures are numbered as they appeared in the RI. Also, data tables presented in the RI are attached to this report on a compact disc.

## **1.1 Site History and Investigation**

This section of the report is largely reproduced from the April 2004 RA. A more detailed discussion of site history and site investigations of the Hookston Station site is presented in the August 2004 Remedial Investigation report (RI) prepared by ERM.

The site is located at the intersection of Hookston and Bancroft Roads in Pleasant Hill, California (RI Figure 1-1). The site covers approximately eight acres, is currently occupied by commercial and light industrial businesses, and is surrounded by a residential neighborhood. RI Figure 1-2 depicts the Hookston Station site.

The site was formerly owned by the Southern Pacific Transportation Company (SPTCo) from June 1891 until September 1983 and was used for a rail line and a station ("Hookston Station"). Between approximately 1965 and 1983, the land was developed into a mixed light industrial business complex. The property was transferred from SPTCo to Mr. Daniel Helix in 1983, and the eastern portion of the site was subsequently purchased by the Contra Costa County Redevelopment Agency (CCCRA) in 1989. Union Pacific merged with SPTCo in 1997 and thereby took over its project responsibilities.

Environmental investigations regarding the presence of chemicals in soil and ground water at the site were initially conducted between 1989 and 1996 by various environmental consulting firms on behalf of Contra Costa County and Mr. Helix. As described below, these investigations discovered the presence of petroleum-based products and chlorinated solvents in the soil and ground water at the site.

The initial environmental investigations by Harding Lawson Associates (HLA, January 1990 and June 1990) were completed for the Contra Costa County Public Works Department in support of the proposed purchase of the eastern portion of the property. Following the discovery of chemical impacts to soil and ground water at the site, Engeo, Inc. (1991 to 1992) and Treadwell & Rollo, Inc. (1993 to 1996) performed additional investigations on behalf of Mr. Helix. UPRR



and Mr. Helix contracted with ERM in April 2000 to perform ground water sampling at the site, build a comprehensive project database, and develop a plan for moving the project through final remediation.

*Modified Phase I Preliminary Report, HLA, 1990*

This October 1989 investigation consisted of the collection of 10 surface soil samples, and focused solely on the presence of petroleum hydrocarbons. As a result of the concentrations of petroleum hydrocarbons found in these samples, HLA recommended a ground water investigation be conducted and additional soil samples be collected.

*Remedial Investigation, HLA, 1990*

This April/May 1990 investigation consisted of the collection of soil and ground water samples. Four monitoring wells (MW-1, MW-2, MW-3 and MW-4) were installed on site and were subsequently sampled for petroleum hydrocarbons. The laboratory contracted for this investigation alerted HLA of the presence of volatile organic compounds (VOCs), particularly trichloroethylene (TCE), in the ground water samples. Shallow soil samples collected during this investigation were not analyzed for VOCs, but HLA recommended additional soil and ground water samples be collected at the site.

*Preliminary Site Characterization, Engeo, 1991*

This investigation consisted of the collection of soil and ground water samples and the installation of two shallow monitoring wells (MW-5 and MW-6). In addition, 76 passive soil vapor probes were installed and analyzed. The soil vapor survey served to locate the areas with elevated VOC concentrations at the site. Based on these findings, Engeo recommended further soil and ground water investigation activities.

*Report on Ground Water Sampling, Engeo, 1992*

This January 1992 investigation consisted of the collection of ground water samples from the six on-site monitoring wells. These samples provided further insight into the extent and concentration of TCE in ground water at the site.

*Initial Soil Characterization Study, Engeo, 1992*

In June 1991, Engeo suggested that a further vertical delineation of VOCs in soil was needed at the site. This January 1992 investigation consisted of the collection of soil and ground water samples to provide an on-site characterization of VOCs. During this investigation, 21 soil borings were advanced and one grab ground water sample was collected. The 1992 report also discusses sanitary sewer video inspections that were performed in three phases between June 1991 and January 1992.

*Subsurface Investigation, Treadwell & Rollo, 1993*

This investigation consisted of the collection of 14 off-site grab ground water samples and the installation of two on-site and two off-site monitoring wells. This was the first off-site investigation; the results indicated that the ground water VOC plume was present up to 2,000 feet downgradient of the site. Three monitoring wells (MW-1D, MW-2D and MW-3D) were installed in the deeper aquifer zone. Sample results from these wells indicated that TCE was also present in the deeper aquifer. This report also identified several off-site private domestic and municipal water wells within the vicinity of the site.

*Supplemental Subsurface Investigation, Treadwell & Rollo, 1996*

This November 1995 field investigation consisted of the collection of soil and ground water samples from numerous on-site and off-site locations. Several shallow on-site soil samples were collected and analyzed for VOCs. In addition to collecting samples from the 10 existing monitoring wells, Treadwell & Rollo advanced 10 shallow HydroPunch borings to further delineate the shallow ground water TCE plume. This report concluded that the increases of PCE in ground water at wells MW-1 and MW-7 may be caused by an off-site, upgradient contaminant source or unknown on-site sources.

*Ground Water Monitoring, ERM, 2000*

In June 2000, Environmental Resources Management (ERM) completed ground water monitoring of nine of the 10 monitoring wells on and downgradient of the site. One of the on-site wells (MW-02) was not sampled due to immobile equipment that blocked access to the wellhead. In September 2000, ERM resampled MW-03D to confirm the elevated detections of TCE reported in the June 2000 laboratory data.

*Preliminary Risk Evaluation (PRE), ERM, 2002*

On October 22, 2002, ERM submitted a Preliminary Risk Evaluation (PRE) for the Hookston Station site to the RWQCB. The PRE was conducted to assess passive exposures to VOCs in ground water underlying the Hookston Station site and nearby neighborhoods.

Development of the PRE focused on the following exposure pathways:

- Inhalation of VOCs released from the ground water table into indoor air;
- Inhalation of VOCs released from the ground water table into outdoor ambient air; and
- Discharge of ground water to the creek, and subsequent exposure by both human and ecological receptors.

Risk-based screening levels were identified or derived for each of these pathways.

To support the PRE, ERM completed surface flux chamber sampling to provide site-specific chemical flux data. These data were used to evaluate potential human health risks associated with vapor migration into indoor air and outdoor ambient air. In addition to the collection of surface flux data, ERM also collected surface water and sediment data to support evaluation of potential human health and ecological risks associated with the discharge of ground water to Walnut Creek.

*Source Area Investigation and Interim Remedial Measures Analysis Report, ERM, 2003*

In November 2003, ERM completed a source area investigation. This report concluded TCE is the primary chemical of concern. Generally low concentrations of TCE were found in soils. The soils are isolated from direct human contact and contact with the underlying ground water. The ground water quality was found to be generally stable. This investigation determined chemical concentrations were not detected at concentrations that warrant consideration for an interim remedial measure (IRM).

Further detail of the overall project background, site history, and previous site characterization results can be found in the *Phase I Remedial Investigation Sampling and Analysis Plan* (Phase I RI SAP) (ERM, 2000) and the *Remedial Investigation Progress Report* (ERM, 2002).

*Remedial Investigation Report, ERM, 2004*

The Remedial Investigation Report (RI) presented the results of the Phase I investigation and the Phase II investigations of the source area and the characterization of ground water. Phase I investigations were conducted for on-site soil vapor (passive methods), on-site and off-site ground water, off-site surface water and sediment in Walnut Creek, and surface flux chamber sampling at both on-site and off-site locations. In addition, residential wells were surveyed in the Hookston Station area.

Phase II investigations of the Hookston Station site evaluated on-site soil and on-site and off-site ground water impacts, soil vapor concentrations of chemicals of potential concern at on-site and off-site locations, and indoor air concentrations of chemicals of potential concern at on-site and off-site locations. These studies completed the dataset necessary to proceed with the Feasibility Study of the Hookston Station site. They also determined that sources of TCE other than the Hookston Station site are impacting area ground water.

## **1.2 Baseline Risk Assessment Objectives and Scope**

The objectives of this baseline RA are to comply with the SFRWQCB order dated August 12, 2004 that requires preparation of a baseline human health RA for the Hookston Station site. The baseline RA presents quantitative estimates of on-site human exposures to chemicals in indoor air and soil and off-site human exposures to indoor air and ground water.

The April 2004 RA presented a screening level evaluation of ground water hypothetically used as a supply of potable water. The baseline RA does not further evaluate potential use of ground water as a potable water supply. Also, possible exposure to chemicals of potential concern in on-site and off-site indoor air that may result from soil or ground water sources are assessed using indoor air data rather than by modeling indoor air concentrations from these possible sources. To evaluate possible contact with chemicals of potential concern in ground water via use for non-potable purposes (i.e., irrigation, filling swimming pools), data from residential wells in the community surrounding Hookston Station are used.

Human health risks resulting from potential exposure to chlorinated ethenes (tetrachloroethylene, trichloroethylene, dichloroethylenes, and vinyl chloride) in indoor air and ground water are calculated for possible Hookston Station sources and Hookston Station plus non-Hookston Station sources. As determined by ERM, the mass of chlorinated ethene compounds contributed by the on-site Hookston Station Site source area to the regional plume is 36%. As such, risks from exposure to chlorinated ethenes are assessed in the baseline RA as being 36% attributable to the Hookston Station site. For the purpose of comparison, the risks resulting from potential exposure to the entire mass of chlorinated ethenes in regional ground water plume are also calculated.

## **2.0 DATA EVALUATION**

This section presents an evaluation of the data used in the baseline RA of the Hookston Station site. The primary source of data for this baseline RA is the RI (ERM, 2004). However, some on-site soil data is used which results from investigations performed in the 1980s and 1990s. All chemicals detected in indoor air, soil, and residential well ground water were retained as chemicals of potential concern in the baseline RA. Data tables from the RI report are attached to the baseline RA on a compact disc.

### **2.1 Summary of Analytical Results**

#### **2.1.1 On-site Indoor Air Sampling Results**

Based on the results of on-site soil vapor sampling, ERM performed on-site indoor air sampling in the office and work space of Hookston Station site businesses in December, 2003. The locations of these samples are presented in Figure 5 from the April 2004 risk assessment. Five indoor air samples (IA-1, IA-2, IA-3, IA-5, and IA-6) were collected at a height of 5 feet using Summa canisters over a period of 8 hours and analyzed for 1,1-dichloroethylene (1,1-DCE), cis-1,2-dichloroethylene (cis-1,2-DCE), and TCE. The volatile organic compounds (VOCs) analyzed in indoor air samples were selected in agreement with the SFRWQCB. Samples IA-1 and IA-6 were collected from enclosed office spaces and samples IA-2, IA-3, and -5 were collected within open warehouse areas. The results of this sampling are presented in Table 2.1.1. In addition, an ambient air sample was collected outdoors at the Hookston Station site (AA-2). 1,1-DCE, cis-1,2-DCE, and TCE were not detected in the ambient air sample at detection limits of  $<0.065$ ,  $<0.13$ , and  $<0.18$   $\mu\text{g}/\text{m}^3$ , respectively.

1,1-DCE was not detected in any on-site indoor air sample (detection limits ranging from  $<0.065$  to  $<0.081$   $\mu\text{g}/\text{m}^3$ ). cis 1,2-DCE was detected in only one sample (1.7  $\mu\text{g}/\text{m}^3$  in IA-2) at a concentration below the RWQCB screening level (10  $\mu\text{g}/\text{m}^3$ ). Trichloroethylene was detected in all five indoor air samples at concentrations ranging from 0.68 to 4.9  $\mu\text{g}/\text{m}^3$ .

#### **2.1.2 On-site Soil Sampling Results**

The locations of on-site soil borings are presented in RI Figure 5-2. Prior to evaluating soil sampling data for use in the baseline RA, the soil data were categorized as shallow soil (less than 3 meters and deep soils) or deeper soil (greater than 3 meters bgs). With the exception of the petroleum hydrocarbon soil analyses from the late 1980s and early 1990s, the results of soil

samples collected from a depth of 0 to 10-feet bgs were considered for use in calculating exposures and risks. In studies conducted before the RI (1989, 1990, and 1992), petroleum hydrocarbon analyses were non-specific and reported analyses for parameters such as “oil and grease. In addition, detection limits for petroleum hydrocarbons in the earliest studies of the Hookston Station site were elevated. For these reasons, only petroleum hydrocarbon analyses from the Remedial Investigation performed by ERM are used in the baseline RA. PCBs were not detected in Hookston Station site soils.

Direct contact with chemicals in deeper soils is much less likely than contact with shallow soils. For several reasons, chemicals detected in shallow soils were used to calculate exposure and risks for on-site workers. In addition to the fact that direct contact or disturbance of chemicals in deeper soils is unlikely, concentrations of the chlorinated ethenes are higher in the shallow soils than in deeper soils (see RI Table 7-3). Also, exposures to chemicals in soil resulting from volatilization of chemicals from soil to indoor air and leaching of chemicals from soil to ground water were addressed in the April 2004 RA. Further, soil samples collected for analysis for semivolatile organic compounds (SVOCs) (RI Table 7-5) and metals (RI Table 7-7) were collected from within the 0 to 10 feet below ground surface (bgs) soil depth range. During the RI, soil samples collected and analyzed for petroleum hydrocarbons as diesel fuel, gasoline, and motor oil were collected at the soil surface or at a depth of 2.5 feet bgs (RI Table 7-4).

A summary of the soil sample results for all chemicals detected in 0 to 10 feet bgs soils at the Hookston Station site is presented in Table 2.1.2. All chemicals detected in shallow soil (less than or equal to 10 feet bgs) were retained as chemicals of potential concern in the baseline RA.

### **2.1.3 Off-site Indoor Air Sampling Results**

Indoor air sampling was conducted by ERM at 16 residences in the Hookston Station area. These residences are located approximately 250 to 1000 feet to the northeast of the Hookston Station site boundary. The 16 residences were sampled during the months of January, February, and March 2004. The indoor air in living spaces in homes and in several cases, air within crawl spaces, was sampled. With the exception of one residence, sampling was conducted during the winter season when indoor air levels would likely be higher than at other times of the year. Each air sample was collected over a period of approximately 12 hours. The dates, times, and locations sampled in each residence, indoor and outdoor temperatures, and indoor percent relative humidity are presented in Table 2.1.3a.

Details concerning the collection of indoor air samples were provided to the RWQCB under separate cover to protect the confidentiality of residents. For this reason, Tables 2.1.3a and 2.1.2b do not list addresses of the residences but identify each residence with a number from 1 to 16 and the street where the residence is located. Generally, residents were approached as possible participants in the indoor air study when the home was located directly over the highest concentrations of the ground water plume. Participation in the indoor air study was entirely voluntary. Thus, the indoor air locations sampled were not under the control of Dan Helix, Union Pacific, ERM, or CTEH.

The results of the indoor air sampling are presented in Table 2.1.3b. 1,1-DCE was infrequently detected in indoor air in levels up to 0.13 ug/m<sup>3</sup>. Cis-1,2-DCE was not detected in indoor air (detection limits 0.12 to 0.14 ug/m<sup>3</sup>). TCE was detected at concentrations up to 5 ug/m<sup>3</sup> in indoor air. Crawl space 1,1-DCE, cis-1,2-DCE, and TCE concentrations were similar to indoor air, ranging up to 0.11, 0.38, and 6.7 ug/m<sup>3</sup>, respectively.

Ambient air samples were collected at two off-site locations. 1,1-DCE and cis-1,2-DCE were not detected in either ambient outdoor air sample (detection limits 0.062 and 0.12 µg/m<sup>3</sup>, respectively). Trichloroethylene (TCE) was detected in one ambient outdoor sample on Thames Drive at a concentration of 0.21 ug/m<sup>3</sup>. Based on the detection of higher concentrations of TCE in indoor air at the same time that the ambient samples were collected, it is unlikely that ambient outdoor air is a significant source of TCE.

#### **2.1.4 Ground Water Sampling Results**

Ground water sampling results from monitoring wells and HydroPunch samplers are summarized in RI report in RI Tables 8-1, 8-2, 8-3, and 8-4 for VOCs, SVOCs, petroleum hydrocarbons, and metals, respectively.

As summarized in the RI report, TCE and its breakdown products such as 1,1-dichloroethylene (1,1-DCE), cis-1,2-dichloroethylene (cis-1,2-DCE), and trans-1,2-dichloroethylene (trans-1,2-DCE) have been detected in ground water on- and off-site to depths of up to 70 feet bgs. A source of TCE impacted ground water is near the southwestern corner of the Hookston Station site. Movement of ground water across the site is toward the north to northeast direction. TCE and its breakdown products have been transported in ground water off-site. Isoconcentration maps of A-Zone ground water depict the extent of tetrachloroethylene (PCE), TCE, cis-1,2-DCE, and 1,1-DCE migration (RI Figures 8-5, 8-6, 8-7, and 8-8, respectively). The A-Zone consists of thin, discontinuous sand stringers found above a depth of about 30 feet bgs. The B-Zone is a

relatively continuous sand interval located between 30 and 70 feet bgs. Isoconcentration maps of the occurrence of PCE, TCE, cis-1,2-DCE, and 1,1-DCE occurrence in B-zone ground water are presented in RI Figures 8-13, 8-14, 8-15, and 8-16, respectively.

Although some residential wells exist at off-site locations, there is no evidence of potable ground water use in the Hookston Station area. However, concentrations of chemicals detected in monitoring wells were screened using conservative SFRWQCB ESLs designed for protection of persons drinking ground water. As summarized in the April 2004 RA, on-site groundwater and off-site ground water near the site exceeds ESLs protective of drinking water (CTEH, 2004). In particular, concentrations of PCE, TCE, and its breakdown products exceed drinking water ESLs.

As discussed in the RI, a private well survey was conducted by ERM beginning in February 2003. Details of the survey are discussed in Section 5.2.2 of the RI report. The private wells identified by ERM for which information was available from homeowners are either not used or are used for landscape irrigation. No use of private well water for drinking water was reported.

ERM also sampled 8 of the private wells in the Hookston Station area. Table 2.1.4a lists information concerning the 8 wells sampled. The wells sampled were on Bermuda Drive, Stimel Drive, Gragg Lane, Thames Drive, and Waterloo Court. To protect the confidentiality of the residents whose wells were sampled, the locations are designated (a) through (h) with only the street name given.

TCE and other chlorinated ethenes were detected in several of the sampled private wells. In addition, very low levels of other chemicals not associated with the Hookston Station site were detected. These included acetone, chloromethane, 1,1-dichloroethane, and 1,2-dichloroethane.

The private well data were selected for evaluation in the baseline RA because these data better represent chemical concentrations in ground water to which off-site residents may be exposed. All chemicals detected in the private wells were retained for evaluation as chemicals of potential concern in the baseline RA.

### **2.1.5 Surface Water Sampling Results**

Walnut Creek, the nearest surface water body to the Hookston Station site, is located about 0.5 mile east of the site. Whether chemicals in off-site ground water affect Walnut Creek is not known. Sampling of Walnut Creek surface water in 2001 and 2002 detected the presence of very low concentrations (less than 5 ug/L) of chlorinated solvents such as cis-1,2-DCE (1.4 ug/L



and lower), PCE (2.6 ug/L and lower), and TCE (3.3 ug/L) (ERM, 2002). The surface water sampling stations in Walnut Creek are presented in RI Figure 9-1. Sampling results are summarized in RI Table 9-1. Although concentrations of toluene were also detected in Walnut Creek at concentrations less than 1 ug/L and methyl tert-butyl ether (MTBE) was detected at a single location in the creek at 8.3 ug/L, these chemicals have not been associated with the Hookston Station site. For this reason, only cis-1,2-DCE, PCE, and TCE were retained as chemicals of potential concern for the baseline RA.

## **2.2 Summary of Chemicals of Potential Concern for Baseline Risk Assessment**

All chemicals detected in on-site air (Table 2.1.1) and on-site soil (Table 2.1.2) were retained as chemicals of potential concern for the baseline RA.

All chemicals detected in off-site residential indoor air samples (Table 2.1.3b) and private well ground water samples (Table 2.1.4b) were retained as chemicals of potential concern for the baseline RA. Cis-1,2-DCE, PCE, and TCE were retained as chemicals of potential concern for surface water in Walnut Creek.

### **3.0 EXPOSURE ASSESSMENT**

The objectives of the exposure assessment are to evaluate potential pathways of human exposure to the chemicals of potential concern in indoor air, soil, ground water, and surface water at or near the Hookston Station site. Once complete exposure pathways are identified (for example, ingestion of a chemical in soil), chemical intakes associated with each pathway are calculated for each potential receptor (such as the construction worker). This section analyzes exposure conditions that may exist on-site at the Hookston Station site as well as conditions that exist off-site in the nearby residential areas.

This exposure assessment calculates chemical intakes for potentially exposed populations that are representative of “reasonable maximum exposure” (RME). The RME is defined by the USEPA as “the highest exposure that is reasonably expected to occur at a site” (USEPA, 1989). The intent of the RME scenario is to calculate chemical intakes that do not underestimate exposure under conservative exposure conditions.

Data used to calculate exposures to chemicals in on-site indoor air and on-site soil are summarized in Tables 2.1.1 and 2.1.2, respectively. Data used to calculate exposures to chemicals in off-site residential indoor air are presented in Table 2.1.3b. Data used to calculate exposures to chemicals in off-site private well water are summarized in Table 2.1.4b. The maximum detected concentrations of cis-1,2-DCE, PCE, and TCE were used to assess possible human exposure to these chemicals in Walnut Creek surface water.

#### **3.1 Exposure Pathway Analysis**

As stated by the USEPA, an exposure pathway “describes the course a chemical or physical agent takes from the source to the exposed individual. An exposure pathway analysis links the sources, locations, and types of environmental releases with population locations and activity patterns to determine the significant pathways of human exposure” (USEPA, 1989).

An exposure pathway is made up of four elements. These are:

- A source and mechanism of chemical release,
- A retention or transport medium,
- A point of potential human contact with the contaminated medium, and;
- An exposure route at the contact point.

In the following discussion, exposure pathways to chemicals in on-site indoor air, on-site soil, off-site indoor air, ground water from private wells, and surface water are identified. These exposure pathways are based on current or reasonable future uses of the Hookston Station site. A summary of potential exposure pathways for on-site and off-site persons is presented in Table 3.1. These potential pathways of exposure are discussed below.

### **3.2 On-site Exposure Pathways**

Due to commercial/industrial land use, on-site workers are the primary receptors of potential concern at the Hookston Station site. Although other individuals (business patrons, visitors) may be exposed by these same pathways on-site, the potential for exposure is small relative to that potentially experienced by on-site workers.

On-site workers may spend time both indoors in offices and work areas and outdoors. For the purpose of assessing exposure to chemicals present in on-site indoor air, workers are conservatively assumed to spend the work day indoors.

Typically, direct contact with chemicals in soil is assumed to occur during outdoor activity, although workers may also contact soil as indoor dust during indoor activity. For this reason, on-site commercial/industrial workers are assumed to have direct contact with surface soil or indoor dust as a part of their normal workdays. However, the likelihood of a commercial/industrial worker directly contacting outdoor surface soil is unlikely since much of the Hookston Station site is covered by base rock or asphalt. Nonetheless, possible direct contact with chemicals in soil is considered for the on-site commercial/industrial worker. Exposures to chemicals of potential concern in soils ranging from surface soil to a depth of 10 feet bgs are assessed for the incidental ingestion, skin contact, and inhalation exposure pathways for the on-site commercial/industrial worker. For this exposure to occur, it would be necessary to remove the overlying base rock and asphalt cover and excavate soils to a depth of 10 feet bgs so that it is brought to the surface.

No disturbance of site soils or other construction activity is planned for the Hookston Station site. However, to address the future possibility of future short-term but intensive exposures to chemicals in subsurface soil, a construction worker soil exposure scenario is considered for the Hookston Station site. A future construction worker is assumed to incidentally ingest chemicals in soil, have skin contact with chemicals in soil, and inhale chemicals of potential concern in soils ranging from the surface to 10 feet bgs.

In summary, the following exposure pathways are assumed to be complete for on-site workers:

#### Commercial/Industrial Workers

- Inhalation of volatile chemicals in indoor air
- Inadvertent ingestion of chemicals in soil
- Skin contact with chemicals in soil
- Inhalation of chemicals in dusts or volatilizing from soil to outdoor air

#### Construction Workers

- Inadvertent ingestion of chemicals in soil
- Skin contact with chemicals in soil
- Inhalation of chemicals in dusts or volatilizing from soil to outdoor air

### **3.3 Off-site Exposure Pathways**

Due to detection of low levels of 1,1-DCE and TCE in off-site indoor residential air, inhalation of these VOCs is considered to be a complete exposure pathway for an adult and child resident.

Private wells have been used in the area of affected off-site ground water and it is possible that ground water from these wells may be used to water lawns and homegrown produce. The VOCs of interest evaporate rapidly from water and are almost completely released from irrigation water into outdoor air (Berisford et al., 2003). Thus, while individuals may be exposed to chemicals volatilizing from irrigation water into outdoor air, it is unlikely that VOCs will be taken up into vegetables to any significant degree. This topic is discussed in greater detail in the uncertainties section of this report. The inhalation of VOCs from ground water used for irrigation is considered a complete exposure pathway for a child and adult resident. Inhalation of VOCs volatilizing from irrigation water from May to September (when water use is highest) is assumed to occur either when the resident is indoors or outdoors.

In addition to irrigation, it is possible that ground water from private wells may be used for cleaning purposes (car washing, etc.), filling swimming pools, and other uses. Of these uses, use of ground water to fill swimming pools would result in the greater amount of exposure to chemicals in ground water. For this reason, recreational exposure to ground water while swimming in a backyard pool is evaluated for a child resident. A child swimmer is assumed to swim 108 times per year (approximately six days per week from May 15 to September 15).

Exposure pathways evaluated for the child swimmer are incidental ingestion, absorption through the skin, and inhalation of volatilizing chemicals while in the pool.

Very low levels of cis-1,2-DCE, PCE, and TCE may be present in Walnut Creek surface water. Direct contact with these chemicals in surface water is unlikely. Exposure and risks posed by these chemicals through fish consumption was addressed in a Preliminary Risk Evaluation (ERM, 2002) and is not further addressed in the baseline RA. In addition, the concentrations of these VOCs are below even the most stringent SFRWQCB surface water ESLs designed to protect surface water.

An exposure pathway not considered in previous risk assessments is volatilization of VOCs from Walnut Creek and inhalation of the VOCs by nearby residents. Several residential properties are adjacent to the creek and it is possible that nearby residents could inhale chemicals volatilizing from the creek. Although this pathway is considered a minor pathway of exposure, it is evaluated quantitatively in the baseline RA.

In summary, the following exposure pathways are assumed to be complete for off-site residents:

Off-site residents (child and adult resident)

- Inhalation of chemicals in indoor air
- Inhalation of chemicals in air released from lawn irrigation with groundwater
- Skin contact, incidental ingestion, and inhalation of chemicals in backyard swimming pools using ground water (child resident only)
- Inhalation of chemicals in air released from Walnut Creek surface water

### **3.3 Quantification of Exposure**

#### **3.3.1 Estimation of Chemical Intakes**

Chemical intakes may be calculated for the on-site and off-site receptors once the concentration of the chemical in air, soil, or ground water is known and the factors associated with human exposure to the medium of concern have been assessed. The 95% upper confidence limit (UCL) on the arithmetic mean soil concentration is typically used to assess reasonable maximum exposures (RME). When the 95% UCL on the arithmetic mean exceeds the maximum

detected concentration, the maximum detected value is often conservatively used to estimate chemical intake (USEPA, 1992). However, due to the location-specific nature of the indoor air results, separate exposures were calculated for each indoor air sample location. In the case of the on-site indoor air results, exposures were calculated for 5 on-site indoor locations. Likewise, exposures were calculated for 11 off-site residences where 1,1-DCE, cis-1,2-DCE, or TCE were detected.

The 95% upper confidence limits (UCLs) on the arithmetic mean concentration for chemicals detected in soil were calculated using the USEPA's ProUCL program (USEPA, 2004). The ProUCL program can be used to calculate UCLs based on several parametric and non-parametric programs. As shown in Table 2.1.2, UCLs were calculated for relatively few chemicals of potential concern. Due to relatively low detection frequencies, calculation of the UCL would not result in a meaningful concentration (due to the large degree of censoring that must be used to evaluate the data). As such, the maximum detected value was often conservatively used as the exposure point concentration (EPC) for calculating soil exposures. The concentration used as the exposure point concentration is bolded in Table 2.1.2. The method used to calculate the UCL (as selected by the ProUCL program) is included in Table 2.1.2.

For the purpose of modeling releases and inhalation exposures to volatile chemicals released from soil, ground water, and surface water, chemicals classified as "volatile" in SFRWQCB guidance (SFRWQCB, 2003) were selected for the development of volatilization factors. Methods and assumptions used to calculate volatilization factors for VOCs released from on-site soil into outdoor air are summarized in Appendix B. Methods and estimated concentrations of chemicals in air resulting from use of ground water for irrigation and to fill a swimming pool are presented in Appendix C. Methods and assumptions used to calculate air concentrations of VOCs resulting from volatilization from Walnut Creek surface water are presented in Appendix D.

Equations and assumptions used to calculate chemical intakes for the on-site commercial/industrial worker inhaling indoor air and the on-site commercial/industrial worker and construction worker exposed to soil are presented in Table 3.2a. Exposure variables used to calculate chemical intakes for off-site residents exposed to VOCs in indoor air, use of ground water for irrigation and filling swimming pools, and VOCs volatilizing from Walnut Creek are presented in Table 3.2b.

Estimates of daily chemical intake are expressed as average daily intakes (ADIs) or lifetime average daily intakes (LADIs). ADIs are calculated over the assumed period of exposure whereas LADIs are calculated over a lifetime (70 years). ADIs and LADIs for ingested and inhaled chemicals of potential concern are expressed as intakes rather than absorbed doses. Dermal contact with chemicals of potential concern in soil and ground water is calculated as an absorbed dose.

ADIs are used to assess noncancer risks whereas LADIs are used to assess lifetime cancer risks.

ADIs were not calculated for lead. Cal-EPA uses the Leadsread exposure model to assess lead exposure and the resulting blood lead concentration resulting from exposure to lead in dust, soil, food, drinking water, and air. Exposure to lead in on-site soil and other environmental media is assessed in Appendix E using the Leadsread 7 model.

ADIs and LADIs for the commercial/industrial worker exposed to VOCs in on-site indoor air are presented in Table 3.3. Soil exposure estimates for the commercial/industrial worker and construction worker are presented in Tables 3.4 and 3.5, respectively.

ADIs and LADIs for off-site residents exposed to VOCs in indoor air, volatilizing from ground water used for irrigation, in swimming pool water, and volatilizing from Walnut Creek surface water are presented in Tables 3.6, 3.7, 3.8, and 3.9, respectively.

## **4.0 TOXICITY ASSESSMENT**

### **4.1 Noncarcinogenic Risks**

The noncarcinogenic effects of the chemicals of concern were assessed by comparing chemical intakes calculated in Section 3 with USEPA reference doses (RfDs). The USEPA considers the RfD to be “an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a portion of the lifetime” (USEPA, 1997b).

The USEPA derives RfDs for inhalation and oral exposure for subchronic exposures (2 weeks to 7 years) and chronic exposures (7 years and longer) for many chemicals. Only chronic inhalation and oral reference doses were used for assessing risks at the Hookston Station site. Inhalation and oral RfDs are presented in Tables 4-1 and 4-2, respectively. RfDs were identified (in order of preference) from the following sources: the Integrated Risk Information Service online, USEPA Region 9 PRG tables (October 2004), and Table J from the SFRWQCB risk assessment guidance (SFRWQCB, 2003).

The RfDs used in this assessment are generally derived from animal studies. The results of these studies are extrapolated to humans using appropriate factors to adjust for uncertainties resulting from:

- Extrapolation from the results of animal studies to humans,
- Variation within individuals of the same species,
- Extrapolation from the results of short-term animal studies and,
- Extrapolation from exposure levels in animal studies that demonstrate an effect rather than a no-effect level.

For any particular chemical, an intake that exceeds the RfD for that chemical indicates that an adverse health effect may be observed. The intake/RfD is defined by the USEPA to be the hazard quotient (HQ) for a chemical. As a general rule, when the  $HQ < 1$ , it is unlikely that an adverse health effect will occur. The chance of observing an effect increases as the HQ increasingly exceeds unity. The USEPA directs that the HQ for each chemical and each route of exposure be summed to calculate a hazard index (HI). This process conservatively assumes that simultaneous exposure to multiple chemicals at intakes below the RfD may produce an adverse health effect if the HI exceeds one. When calculated according to USEPA methods, the HI assumes that the effects of each chemical are additive. The HI is used as a screen to



determine whether or not the effects of intake of multiple chemicals may be of concern. If the HI is less than one, there is little reason to expect that any adverse effect will result from concurrent exposure to all of the chemicals of concern.

The USEPA does not derive dermal RfDs for chemicals. However, since dermal exposure may add to the overall intake of a chemical and possibly cause an adverse effect, the oral RfD was used as the dermal reference dose.

## 4.2 Carcinogenic Risks

The chemicals detected in indoor air, soil and ground water that are considered by the USEPA and the Cal-EPA to be potentially carcinogenic to humans are presented in Tables 4.1 and 4.2. According to the current USEPA weight of evidence classification scheme, Group A chemicals are considered to be human carcinogens. Group B chemicals are considered to be “probable human carcinogens” primarily based on cancer studies in animals. Chemicals in Group B1 have limited evidence of carcinogenicity in humans while Group B2 chemicals are described as having sufficient evidence of carcinogenicity in animals with inadequate human evidence. Chemicals in Group C are considered “possible human carcinogens” on the basis of limited evidence of carcinogenicity in animals.

The Toxicity Criteria Database (<http://www.oehha.ca.gov/risk/ChemicalDB/index.asp>) maintained by Cal/EPA and OEHHA was used as the source of slope factors for the potentially carcinogenic chemicals of potential concern. Cal/EPA and USEPA calculate slope factors by applying the linearized multistage or linear model to data from animal carcinogenicity studies or human epidemiological studies. In the absence of data concerning the carcinogenic potential of very low doses of a chemical, these models are used to generate estimates of carcinogenic potency. An inherent conservatism in these models is the provision that there is no dose, no matter how small, that is not associated with some carcinogenic risk. The uncertainties associated with weight-of-evidence classifications and use of the linear and linearized multistage model are addressed in a later section of this report. Multiplication of the lifetime average daily intake by the slope factor [expressed as (mg/kg/day)<sup>-1</sup>] produces a unitless estimate of lifetime cancer risk. Increased lifetime cancer risk calculated by this method is often expressed in terms of 1 in ten thousand (1E-04), 1 in one hundred thousand (1E-05), or 1 in one million (1E-06).

In cases where both the USEPA and DTSC have derived different slope factors for the same chemical, the DTSC slope factor was used.

### **4.3 Toxicological Effects of Lead**

Unlike other chemicals for which human exposure is calculated in terms of chemical intake (intake in milligrams of chemical per kilogram of body weight per day, mg/kg/day), risks associated with exposure to lead are based on blood lead concentrations. Due to the existence of an ever-growing database relating blood lead concentration (typically expressed in terms of micrograms of lead per deciliter of blood,  $\mu\text{g/dL}$ ) and human toxicity, blood lead concentration is the most direct means by which the toxic effects of lead in humans can be assessed.

The USEPA and others have developed lead exposure models for evaluating blood lead concentrations associated with intake of lead from food, water, air, and soil. The State of California has developed its own lead exposure model to calculate lead exposure in children and adults. The DTSC child and adult lead exposure models were used to calculate blood lead concentrations for workers potentially exposed to lead in soil at the Hookston Station site. These calculations are presented in Appendix E.

### **4.4 Toxicological Effects of Petroleum Hydrocarbon Mixtures**

There is currently no single, universally accepted method for addressing risks posed by petroleum hydrocarbon mixtures in soil or water. For example, although petroleum mixtures in soil at the Hookston Station site were analyzed as “diesel”, “gasoline”, and “motor oil”, there is no reference dose available for estimating the toxicity of these mixtures. The problems associated with the evaluation of risks associated petroleum mixtures in the environment relate to the analytical characterization of petroleum mixtures, the uncertainties associated with a relative lack of toxicological information concerning the toxicity of whole petroleum mixtures, and the effect of weathering on petroleum mixtures in the environment. In the absence of further analytical characterization of the petroleum hydrocarbon fractions in soil, the SFRWQCB conservatively assumes that the petroleum hydrocarbons are the most toxic petroleum hydrocarbon fraction.

## **5.0 RISK CHARACTERIZATION**

The risk characterization portion of the risk assessment integrates the results of the exposure assessment (Section 3) and toxicity assessment (Section 4) to calculate theoretical estimates of noncancer and lifetime cancer risks. In addition, uncertainties associated with the baseline RA are discussed.

Calculated noncancer and theoretical lifetime cancer risks for individual chemicals are summed for each exposure pathway. In addition, summed risks for each exposure pathway are added together to calculate a cumulative risk calculation for each exposure scenario.

Noncancer and theoretical lifetime cancer risks resulting from commercial/industrial worker exposure to on-site indoor air and on-site soil are presented in Tables 5.1 and 5.2, respectively. Risks calculated for the on-site construction worker exposed to soil are presented in Table 5.3.

Noncancer and theoretical lifetime cancer risks for the off-site resident exposed to VOCs in indoor air, VOCs volatilizing from ground water used for irrigation, resident swimming exposure to VOCs in ground water used to fill a pool, and VOCs volatilizing from Walnut Creek surface water are presented in Tables 5.4, 5.5, 5.6, and 5.7, respectively. Noncancer and theoretical lifetime cancer risks calculated for residents exposed to chlorinated ethenes (1,1-DCE, cis-1,2-DCE, trans-1,2-DCE, TCE, and vinyl chloride) are allocated to Hookston Station sources (accounting for a 0.36 fraction of the mass of chlorinated ethenes in indoor air, ground water, and surface water) and the total from all sources (on-site Hookston Station and non-Hookston Station sources).

These risks are discussed for on-site and off-site receptors below.

### **5.1 On-Site Exposure to Chemicals in Indoor Air and Soil**

Noncancer and theoretical lifetime cancer risks calculated for the commercial/industrial worker exposed to cis-1,2-DCE and TCE in indoor air are presented in Table 5.1. The summed hazard quotients (noncancer risks) were less than one for each on-site indoor air location sampled, indicating that inhalation of on-site indoor air would not result in adverse noncancer health effects. Theoretical lifetime cancer risks associated with inhalation of TCE ranged from 3.3E-07 at location IA-3 to 2.4 E-06 at location IA-2. These risks are well within the range of risks considered to be acceptably low by the USEPA (i.e., 1E-06 to 1E-04) and below the one in 100,000 (1E-05) theoretical lifetime cancer risk level considered to pose “no significant risk” as defined under the State of California’s Proposition 65.

Noncancer and theoretical lifetime cancer risks calculated for the commercial/industrial worker and construction worker exposed to chemicals of potential concern in soil are presented in Tables 5.2 and 5.3, respectively. Summed noncancer risks for the commercial/industrial worker and construction worker exposed to the chemicals of potential concern in soil were below one, indicating that exposure to chemicals in soil would not result in noncancer health risks to on-site workers. As presented in Appendix E, commercial/industrial worker and construction workers exposed to 104 mg/kg lead in soil would be unlikely to have a blood lead concentration greater than 10 ug/dL, the targeted level of concern for children and women of child-bearing age.

Theoretical lifetime cancer risks for the commercial/industrial worker exposed to chemicals of potential concern in soil were  $3.1 \times 10^{-4}$  and exceeded the range of risks targeted by the USEPA ( $1 \times 10^{-6}$  to  $1 \times 10^{-4}$ ). Arsenic accounts for 98% of the total lifetime cancer risk calculated and no other chemical exceeded a theoretical lifetime cancer risk of  $1 \times 10^{-5}$ . Since publication of the April 2004 RA, OEHHA changed the oral slope factor for arsenic, increasing its carcinogenic potency approximately 6-fold. In addition, the exposure point concentration for arsenic in soil (132 mg/kg) is skewed relatively high on the basis of two samples. Soil samples B-69 (211 mg/kg) and B-84 (76 mg/kg) were collected at locations approximately 500 feet apart on the Hookston Station site. The remaining arsenic soil concentrations are below 10 mg/kg, suggesting that the occurrences of elevated concentrations in surface soils are anomalous. Also, recent evidence indicates that a substantial fraction of arsenic in soil that is ingested may not be absorbed from the gastrointestinal tract. This baseline RA assumes the conservative default condition, i.e., that arsenic in soil is absorbed from the gastrointestinal tract to the same degree as arsenic dissolved in water. The risk assessment uncertainties associated with assessing arsenic bioavailability and risk are discussed in Section 5.3 below.

Theoretical lifetime cancer risks calculated for the construction worker exposed to chemicals of potential concern in soil were  $4.3 \times 10^{-5}$ . This risk is within the range of theoretical lifetime cancer risks considered acceptable by the USEPA. As with the commercial/industrial worker, the risk is almost entirely attributable to arsenic.

## **5.2 Off-Site Exposure to Chemicals in Indoor Air, Ground Water, and Surface Water**

Risks to both off-site child and adult residents were calculated. Noncancer risks are based on exposures calculated for children so as not to underestimate noncancer risks. Theoretical lifetime cancer risks for residents are the sum of the child and adult lifetime cancer risks.

Noncancer risks calculated for the off-site resident exposed to VOCs in indoor air (Table 5.4),

VOCs volatilizing from ground water used for irrigation (Table 5.5), VOCs in ground water used to fill a swimming pool (Table 5.6), and VOCs volatilizing from Walnut Creek (Table 5.7) were less than one even when the risks from Hookston and non-Hookston sources were combined. This indicates that VOCs detected in residential indoor air, ground water from private wells, and surface water from Walnut Creek do not pose noncancer adverse health risks.

All theoretical lifetime cancer risks calculated for off-site residents were within the acceptable USEPA risk range ( $1\text{E-}06$  to  $1\text{E-}04$ ) and are below the “no significant risk” level for carcinogens of  $1\text{E-}05$  defined under Proposition 65. The highest off-site theoretical lifetime cancer risk calculated was for a resident exposed to VOCs in indoor air (4. Hampton Drive; Table 5.4). Risk allocated to the Hookston Station site for this residence was  $1.4\text{ E-}06$ . Even when risks from Hookston Station plus non-Hookston Station sources are considered, the highest risk calculated is  $3.9\text{ E-}06$  (4. Hampton Drive; Table 5.4).

The highest theoretical lifetime cancer risk calculated for the irrigation exposure scenario was calculated for a private well on Bermuda Drive ((a) Bermuda) (Table 5.5). Risks allocated to possible sources of chlorinated ethenes from the Hookston Station site were  $1.3\text{ E-}07$ . Even when all sources of chlorinated ethenes were considered (Hookston Station plus non-Hookston Station sources), the theoretical lifetime cancer risk was  $3.5\text{ E-}07$ , well below the range of risks considered acceptable by the USEPA.

The calculated theoretical lifetime cancer risk for the swimming pool exposure scenario was highest for the dermal absorption pathway. As in the irrigation exposure scenario, the highest theoretical lifetime cancer risk was calculated for a private well on Bermuda Drive ((a) Bermuda) (Table 5.6). Risks allocated to possible sources of chlorinated ethenes from the Hookston Station site were  $2.3\text{ E-}07$ . Even when all sources of chlorinated ethenes were considered (Hookston Station plus non-Hookston Station sources), the theoretical lifetime cancer risk was  $6.3\text{ E-}07$ , well below the range of risks considered acceptable by the USEPA.

Calculated theoretical lifetime cancer risk for residents exposed to VOCs volatilizing from surface water were  $1.6\text{ E-}06$ , well within the range of risks considered acceptable the USEPA. The exposures and risks calculated for this pathway are particularly conservative since changes in wind direction and distance from the creek are not reflected in the calculations. Further, maximum detected concentrations of cis-1,2-DCE, PCE, and TCE in Walnut Creek were used to calculate exposure and risks.

### **5.3 Evaluation of Risk Assessment Uncertainties**

Several areas of uncertainty were associated with the estimation of chemical intakes from exposure to air, soil, ground water, and surface water and the characterization of risk. For ease of discussion, uncertainties are discussed as they relate to either the estimation of exposure or the evaluation of chemical toxicity.

#### **5.3.1 Uncertainties Related to Estimation of Exposure**

Uncertainties associated with estimation of exposure to the chemicals of concern in soil, ground water, or air primarily relate to:

- the representativeness of indoor air samples collected on- and off-site and the possible changes in indoor air concentrations with the season;
- the modeled air concentrations of VOCs associated with use of ground water for irrigation and volatilization of VOCs from surface water;
- the selection of exposure parameters estimating intakes and frequency and duration of exposure;
- the extent of absorption of chemicals in soil from the digestive tract,
- the possible uptake of VOCs into edible vegetable produce grown in home gardens that are irrigated with ground water

These areas of uncertainty are discussed below.

#### Indoor Air Uncertainties

Indoor air samples were collected at on-site businesses in December 2003 and in off-site residences in January, February, and March, 2004. The results represent a single air sample collected on a particular day and may not represent indoor air concentrations throughout the year. The concentrations of VOCs in indoor air can be affected by several factors. When a subsurface source of VOCs is considered (i.e., VOCs in ground water), the most important factors that may affect the migration of VOCs from a subsurface source to the indoor air are the depth of the source of VOCs (i.e., the depth of potentially affected ground water), the permeability of nearby soil and the residence to vapors, and the amount of under-pressurization of the residence. Each of these factors may be directly or indirectly influenced by local climate and meteorological conditions.

In winter, there is typically lower air exchange between the indoor and outdoor environment, resulting in relatively higher VOC concentrations in indoor air than those present in summer. In addition, operation of a heating system produces temperature and pressure differences

between the indoor and outdoor environments that may draw VOCs into a building. The pressure difference inside and outside of a building is greatest when windows and doors are closed and the heating system is operating. Operation of heating systems may create a “stack” effect in the home in which make-up air is pulled into the home at lower levels, potentially drawing vapors from soil into indoor air. Steady winds on a structure at speeds greater than five miles per hour may also cause under pressurization. Thus, the winter heating season and steady winds may create conditions that increase movement of soil vapor into a home.

In addition, during the wetter seasons of the year, precipitation may fill pores occupied by soil vapor, driving the soil vapor from the wetter soils into the drier soils underneath a home. The shallow ground water may also rise during the wetter seasons of the year and bring VOCs in the water table closer to the building.

As a result of these meteorological and climatological conditions, “worst-case” indoor air concentrations may occur during winter or spring when depth to groundwater is shallow, the building heating system is operating, and the doors and windows of the building are closed (MADEP, 2002). Although the Pleasant Hill/Concord area experiences milder winters than most areas of the country, indoor air concentrations of VOCs measured in December through March in the Pleasant Hill/Concord area are expected to be higher than in the summer and fall months for the reasons described above. Therefore, the results of winter-time indoor air sampling conducted at on- and off-site areas at Hookston Station may overestimate year-round average indoor air concentrations.

Crawl space air samples results from 7 off-site residences provide a conservative upper bound for indoor air concentrations observed in residences. The maximum detected concentration of TCE in Hookston Station area crawl spaces ( $6.7 \text{ ug/m}^3$ ) was very similar to the maximum TCE concentration detected in residential indoor air ( $5 \text{ ug/m}^3$ ) (Table 2.1.3b). According to studies of radon migration into residential indoor air, indoor air concentrations of radon ranged from 0.36 to 0.60 of crawl space radon concentrations (Nazaroff and Doyle, 1985). This suggests that crawl space air concentrations of VOCs would likely exceed the concentration of VOCs in indoor air if the crawl space is the source of VOCs migrating into the residence. The observation that residential indoor air VOC concentrations are similar to crawl space air concentrations suggests that the indoor air sampling program has not underestimated the concentration of VOCs migrating into the indoor air of living spaces.

With exception of a voluntary indoor air questionnaire, the possible sources of VOCs in indoor air were not thoroughly investigated. Although migration of soil vapor from ground water may

be a source of VOCs in indoor air, indoor use of consumer products containing VOCs may also be a source of VOCs in indoor air. The degree to which other sources of VOCs such as consumer products may be sources of VOCs in indoor air is not known. For example, the Agency for Toxic Substances and Disease Registry estimated that "typical" background concentrations of TCE in air ranged from about 0.5 ug/m<sup>3</sup> to 2.7 ug/m<sup>3</sup> (ATSDR, 1997). CalEPA estimated that the median range of TCE concentrations in California homes ranged from 0.3 ug/m<sup>3</sup> to 0.8 ug/m<sup>3</sup> (CalEPA, 2001b). Thus, it would not be surprising to find TCE in indoor air in a city or suburban area at typical levels that approach or even exceed the RWQCB's residential indoor air screening level of 1.2 ug/m<sup>3</sup>.

Concentrations of TCE in residential indoor air are similar to what would be predicted from empirically derived attenuation factors for VOCs in soil vapor and indoor air. Johnson et al. (2002) examined the relationship between soil gas and indoor air concentrations of several chlorinated VOCs. The range of observed attenuation factors between soil vapor and indoor air was 1E-4 to 1E-6 with an average of 3 E-05. Given the range of attenuation factors reported by Johnson et al. and the maximum TCE soil vapor concentration detected off-site near the Hookston Station site (ASV-05; 6,800 ug/m<sup>3</sup>) (RI Table 7-2; RI Figure 5-6), indoor air concentrations would be predicted to range from 0.0068 to 0.68 ug/m<sup>3</sup>. These concentrations are somewhat lower than the observed concentrations in indoor air in off-site residences.

#### Air Emissions and Modeling Uncertainties

Air concentrations resulting from modeling use of ground water for irrigation purposes, filling pools, and VOC emissions from surface water are uncertain. To some degree, the potential for human exposure to volatile emissions is maximized by the fact that wind direction is not factored into the analysis. It is highly unlikely that the wind carrying VOC emissions is toward the home of residents 100% of the time. Also, more rapid volatilization of VOCs from irrigation water or swimming pools could result in decreased inhalation exposures. For example, splashing in a pool could result in greater than expected losses of VOCs from surface water.

#### Exposure Parameters

Exposure periods of 30 years are routinely considered in risk assessments. As is typical with risk assessments, no factor was used to account for the likely decrease in ground water concentrations over time. Over decades, concentrations of chemicals of potential concern in ground water will likely decrease due to degradation, volatilization, or other mechanisms. Assumption of a constant concentration of the chemicals of potential concern in ground water will likely result in overestimation of chemical exposures and risks.



Indoor air inhalation rates were adjusted downward from the default daily inhalation rates for off-site residents exposed to VOCs in indoor air. A daily indoor inhalation rate of 13.3 m<sup>3</sup>/day was used for the adult resident (average of the male and female average daily inhalation rates) and 8.7 m<sup>3</sup>/day used for the 1 to 7 year old child. These rates are average values recommended for use by the USEPA (USEPA, 1997a) for daily inhalation exposure. Inhalation rates less than 20 m<sup>3</sup>/day for adults and 10 m<sup>3</sup>/day for children are justified for indoor air exposures, particularly when it is considered that USEPA recommends that the amount of time spent indoors at a residence is 16.4 hours per day (USEPA, 1997a).

#### Bioavailability of Chemicals in Soil

The absorption of chemicals in soil from the digestive tract has been the subject of considerable study in recent years. In the calculation of ESLs for direct contact exposure scenarios, the regulatory default is to assume that ingestion of a chemical in soil is absorbed to the same extent as the chemical in food or water. For arsenic, this is clearly not the case. Roberts et al. 2002 evaluated the digestive tract absorption of arsenic in monkeys for soils from five waste sites (one soil sample from an electrical substation, a wood preservative treatment site, and a cattle-dip vat site and two samples from pesticide sites) with arsenic concentrations in soil ranging from 101 to 743 mg/kg arsenic. The absorption of arsenic in soil was measured relative to that of an oral solution of sodium arsenate. The excretion of urinary and fecal arsenic was used to evaluate the bioavailability of arsenic. The monkeys received oral doses of arsenic in soil ranging from 0.3 mg/kg to 1.0 mg/kg. The oral bioavailability of arsenic in soil (relative to sodium arsenate administered orally in water) ranged from 10.7% (pesticide site soil) to 24.7% (cattle dip site soil). Roberts et al. concluded that "These observations, coupled with data in the literature, suggest limited oral bioavailability of arsenic in soils from a variety of types of arsenic-contaminated sites." In summary, these data sets suggest that arsenic bioavailability in soil and dust is considerably lower than 100%. For this reason, theoretical risks calculated to be associated with direct contact with arsenic in soil are likely overestimated by a factor of about 3.

#### Uptake of VOCs into Homegrown Produce

Chemicals detected in ground water at the Hookston Station site are volatile, meaning that they evaporate easily at normal temperatures. As a result, VOCs will tend to volatilize during the irrigation process, rather than be taken up or absorbed by plants. Berisford et al. 2003 demonstrated that TCE and tetrachloroethylene (PCE) are readily stripped from ground water when sprayed through mini-sprinklers. These sprinklers, typical of home and garden use, stripped concentrations of TCE and PCE from ground water with an effectiveness of 97% to 100%. Berisford et al. (2003) tested concentrations of PCE and TCE that ranged from hundreds to thousands of ug/L. For this reason, it is likely that lawn or garden irrigation will result primarily

in releases of VOCs to air but will not result in significant transfer of VOCs to edible fruits and vegetables. The possible exposure of off-site residents to VOCs in air resulting from lawn irrigation is addressed in Appendix C.

Research demonstrates that if the volatile chemicals manage to reach the plants and if the chemicals are then absorbed by the plants, the VOCs do not accumulate in plant tissues (Davis et al. 1998). Instead, the VOCs are transferred to air through pores in the plants' tissues. The resulting air concentrations do not pose a threat to health because the amounts of chemicals released are very low and they mix readily with surrounding air.

Studies have also shown that chemicals taken up through a plant's root system tend to concentrate in the cells near the surface of the roots (Agustin 1994). In root vegetables such as beets, carrots, and potatoes, these cells are typically lost during washing and peeling of the produce. In above-ground fruits and vegetables (e.g., tomatoes, lettuce, squash, etc.), the roots are not consumed.

Plants are also able to break down or degrade volatile chemicals. Consequently, volatile chemicals taken up by plants may be present temporarily in the roots and stems of the plant, but are much less likely to be present in the leaves or other above-ground, potentially edible parts of the plant (Newman et al. 1997). According to research performed by Schnabel et al. (1997), TCE that is taken up into tomatoes and spinach is degraded to the extent that TCE is not detectable. Schnabel et al. were unable to remove the breakdown products of TCE using a strong solvent or acid, indicating that if the breakdown products in tomatoes or spinach are eaten, it is unlikely that they would be absorbed from the digestive tract. In summary, the literature review indicates that uptake and accumulation of volatile chemicals in plants and subsequent exposures by home gardeners and their families are likely to be negligible.

### **5.3.2 Uncertainties Related to the Toxicity Assessment**

Uncertainties associated with characterization of risks associated with the chemicals of concern primarily relate to the derivation of cancer slope factors and their use in estimating lifetime cancer risk. Perhaps the greatest uncertainty associated with the risk assessment process is the evaluation of carcinogenic risk due to chemical exposure. The fundamental principles underlying risk assessment for carcinogenic chemicals remain arguable, including the tenet that every potential carcinogen is associated with some degree of carcinogenic risk, no matter how small the dose. The belief that chemically induced cancer is a non-threshold process is a conservative default policy that the EPA assumes to ensure the protection of human health.

However, there is little biological basis to support the widespread application of this policy to all potential carcinogens.

The EPA default policy for potential chemical carcinogens mandates that results from high-dose animal studies be extrapolated to exposures in humans which are thousands of times lower. The EPA uses a mathematical model known as the linearized multistage model to extrapolate from high doses to very low doses. As applied by the EPA, the linearized multistage model leads to quantitative estimates of cancer risk which are conservative, upper bound approximations of lifetime cancer risk. The EPA expressed the following uncertainty in using the linearized multistage model to determine carcinogenic risks in humans:

It should be emphasized that the linearized multistage procedure leads to a plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true value of risk is unknown, and may be as low as zero. The range of risks, defined by the upper limit given by the chosen model and the lower limit which may be stated as low as zero, should be explicitly stated. (51 Federal Register 33998)

Thus, according to the EPA commentary cited above, carcinogenic risks estimated using the linearized multistage procedure lead to conservative but not necessarily realistic estimates of risk. The National Research Council has also commented concerning use of the linearized multistage model, stating:

The linearized multistage model is widely used to estimate cancer risks associated with environmental exposures (EPA, 1987) and is said to provide an upper-limit estimate of low-dose response. To some degree, the model's wide use reflects its mathematical flexibility. However, biologic support for the assumption of linearity at low doses remains largely inferential and probably wrong in a high proportion of cases (emphasis added) (Bailar et al., 1988). (NRC, 1989)

For these reasons, it is likely that the risks calculated in this report will substantially overestimate the actual risks which may be associated with exposure to the chemicals of potential concern in air, soil, ground water, and surface water.

In particular, TCE is regulated by the USEPA and State of California as a potentially carcinogenic substance. TCE is also a chemical of concern at the Hookston Station site. In 2001, the USEPA published a draft evaluation of the toxicity of TCE. Although this document has undergone review by the USEPA's Science Advisory Board, a final version of the report has

not been published three years later. Some elements of the draft report are controversial, and apparently the USEPA has asked the National Academy of Sciences to review the report (<http://www.hsia.org/updates/nov-dec%202003.htm>). The uncertainties associated with assessing the theoretical risks of TCE exposure are addressed in greater detail below.

The 2001 draft USEPA toxicity assessment for TCE proposes that TCE should be considered a more potent potential carcinogen than was previously thought by the USEPA or as is currently considered by the Office of Environmental Health Hazard Assessment (OEHHA) in California. Unlike its previous policy regarding the calculation of TCE cancer risk, the USEPA draft report does not propose a single value for assessing TCE cancer risk, but provides a range of values that varies over 20-fold. Each value is based on endpoints derived from a different human or animal study. The draft report recommends that a slope factor from this range be selected that is appropriate for the risk assessment and exposure scenario under consideration, but does not provide specific guidance on choosing an appropriate factor. Publication of the EPA's draft assessment has generated substantial controversy and criticism. In fact, the EPA's own Scientific Advisory Board (SAB) has suggested that risk assessors wait for resolution of controversial issues before putting the new recommendations into effect. The EPA plans to submit the TCE draft assessment to the National Academy of Sciences for a special expert-panel review. That review is scheduled to be completed in 2006.

Concerns raised during review of the USEPA TCE risk assessment draft included:

- Proper use of the range of slope factors developed
- Inadequate scientific analyses to support the range of values
- Oral to inhalation extrapolation
- Risk assessment at background values
- The inconsistency in the human data for the cancer causing effects of TCE

The new range of slope factors for TCE proposed in the USEPA draft assessment is 0.02 to 0.4 per mg/kg-day. The highest value (0.4 per mg/kg-day) is 36-fold higher than the value applied by EPA and 57 times greater than the value derived by the State of California Office of Human Health and Environmental Assessment (OEHHA). A number of problems are associated with using the proposed draft range of values.

- There are no guidelines for applying the range in risk assessment.
- All slope factors are given in terms of oral exposure with no specific guidance for extrapolating from oral to inhalation exposures.

- The high end of the USEPA draft range is based on an inappropriate study.

First, the USEPA draft recommends choosing an appropriate slope factor from the new range based on the unique risk factors of each individual risk assessment. However, the USEPA provides no clear guidance on how this should be done.

Second, the draft slope factors are expressed in terms of oral exposure (per mg/kg-day) without specific information on how to assess inhalation exposures using these values. As a result, federal and state agencies have proposed different methods to address this issue, but there is no consensus. Application of the most conservative slope factor results in an inhalation risk that is many-fold higher than the previous value.

Thirdly, the draft high end slope factor (0.4 per mg/kg-day) calculated by the USEPA was based on the results of Cohn et al. (1994). The Cohn study was a proportional mortality (PMR) study that evaluated the incidence of leukemia and non-Hodgkin's lymphoma (NHL) in a population exposed to solvents in drinking water. In general, PMR studies are unreliable in proving an association, cannot be used to prove causation (McLaughlin and Brookmeyer, 1994) and are entirely inappropriate for deriving health-based benchmarks. Although the study reported small, statistically significant increases in NHL in females exposed to the highest levels of TCE, the study is flawed, causing the relatively small increases in relative risk to be suspect. The study provides no information regarding: 1) residential history of the individuals that describes how long they were exposed; 2) the magnitude of TCE exposure; 3) potential confounders for attributing the increased risk of cancer to TCE; 4) medical history of the residents and; 5) exposure to other potentially carcinogenic substances.

The incompatibility of the draft high end slope factor with empirical evidence of TCE carcinogenicity is illustrated by using the draft slope factor to calculate the risks from workplace exposure to TCE. The most highly exposed individuals are those who have worked with TCE for decades and have been exposed at levels that approach or even exceed current occupational exposure limits. The current Occupational Safety and Health Administration Permissible Exposure Limit (OSHA PEL) for TCE is 537 mg/m<sup>3</sup> and the current American Conference of Governmental Industrial Hygienists Time-Weighted Average Threshold-Limit Value (ACGIH TWA-TLV) for an eight hour work day is 269 mg/m<sup>3</sup>.

Using the most conservative draft slope factor in the range of values derived by USEPA (0.4 mg/kg-day<sup>-1</sup>), 98% or more of all individuals exposed to TCE at current occupational exposure limits would be expected to develop cancer after exposure for 250 days per year for 25 years.

This is clearly not the case. Workers have been exposed to TCE at similar levels for over 80 years and yet no conclusive association between TCE and cancer has been determined, much less one of such magnitude. This level of calculated theoretical risk is unrealistically high, particularly when compared to the results of epidemiological data from TCE exposed workers.

In summary, if TCE exposure in workers caused cancer at the levels suggested by calculations using the EPA's draft high end slope factor, the epidemiological data from exposed workers should indicate a clearly elevated, consistent risk of developing cancer (above that which occurs naturally) after an appropriate latency period. The use of the draft high end value is clearly not supported by the scientific literature.

A review of uncertainties associated with the characterization of human health risk posed by exposure to chemicals in air, soil, ground water, and surface water on or near the Hookston Station site indicates that the methods used primarily overestimate exposure and risk.

## 6.0 RISK ASSESSMENT SUMMARY

The purpose of this baseline RA was to perform an exposure and risk assessment of persons on and near the Hookston Station site that may be exposed to chemicals in indoor air, on-site soil, ground water from off-site private wells, and surface water in Walnut Creek. The baseline RA uses modeling performed by ERM to determine risks to chlorinated ethenes that may emanate from Hookston Station and allocate risks to on-site Hookston Station related sources of contaminants. The ERM modeling indicates that 36% of chlorinated ethenes in the regional ground water plume may result from on-site Hookston Station sources. This percentage was used to allocate risk from on-site Hookston Station sources to off-site receptors. In addition, noncancer and theoretical lifetime cancer risk calculations that account for the sum of Hookston-related as well as non-Hookston related chlorinated ethenes.

Like all risk assessments of this type, the results cannot be used to accurately predict the actual incidence of human disease for current or future conditions. Risks calculated in the baseline RA rely on conservative but uncertain methods.

The RI report was the source of all indoor air, soil, ground water, and surface water data used in evaluating human exposures and risks.

### On-site Exposures and Risks

Evaluation of potentially exposed individuals and possible exposure pathways resulted in selection of the following on-site exposure pathways for consideration in the baseline RA:

#### *Commercial/Industrial Workers*

- Inhalation of volatile chemicals in indoor air
- Inadvertent ingestion of chemicals in soil
- Skin contact with chemicals in soil
- Inhalation of chemicals in dusts or volatilizing from soil to outdoor air

#### *Construction Workers*

- Inadvertent ingestion of chemicals in soil
- Skin contact with chemicals in soil
- Inhalation of chemicals in dusts or volatilizing from soil to outdoor air

Noncancer risks resulting from commercial/industrial worker to VOCs in indoor air were below one for all 5 on-site indoor air locations sampled, indicating that VOCs in indoor air do not pose a noncancer health risk to workers. Theoretical lifetime cancer risk estimates for

commercial/industrial worker inhalation of TCE in indoor air ranged from  $3.3 \times 10^{-7}$  to  $2.4 \times 10^{-6}$ , below or well-within the range of risks considered acceptable by the USEPA.

Noncancer risks resulting from commercial/industrial worker exposure to chemicals of potential concern in soils to a depth of 0 to 10 feet bgs were also acceptably low (0.55). Theoretical lifetime cancer risks exceeded  $1 \times 10^{-4}$ . The lifetime cancer risk associated with commercial/industrial worker exposure to chemicals in soil was 98% attributable to arsenic. Although arsenic concentrations in 17 out of 19 surface soil samples were below 10 mg/kg, two samples were sufficiently high (211 mg/kg and 76 mg/kg) to skew the exposure point concentration to 132 mg/kg for arsenic in on-site soils. Furthermore, much of the site is covered with rock or asphalt, decreasing the possibility that on-site workers will directly contact arsenic in soil.

Noncancer risks for construction workers ingesting soil, having skin contact with soil, and inhaling chemicals in soil was 0.30, indicating that direct contact with on-site soil does not pose a noncancer health concern for construction workers. The lifetime cancer risk associated with construction worker exposure to chemicals in on-site soil was  $4.3 \times 10^{-5}$ , within the range of acceptable USEPA lifetime cancer risks.

#### Off-Site Exposure and Risks

Evaluation of potentially exposed individuals and possible exposure pathways resulted in selection of the following off-site exposure pathways for consideration in the baseline RA:

##### *Off-site residents (child and adult resident)*

- Inhalation of chemicals in indoor air
- Inhalation of chemicals in outdoor/indoor air released from lawn irrigation with groundwater
- Skin contact, incidental ingestion, and inhalation of chemicals in backyard swimming pools using ground water (child resident only)
- Inhalation of chemicals in outdoor/indoor air released from Walnut Creek surface water

Noncancer risks resulting from off-site resident exposure to VOCs in indoor air, VOCs volatilizing from ground water used for irrigation, swimming in ground water used to fill a pool,



and inhalation of VOCs released from Walnut Creek were below one, indicating that these exposures would not result in noncancer health risks.

Theoretical lifetime cancer risks for the off-site resident exposed to VOCs in indoor air varied with the residential location sampled; the range of theoretical lifetime cancer risk for risk attributable to on-site Hookston Station sources was  $5.3 \text{ E-}08$  to  $1.4 \text{ E-}06$ . The range of theoretical lifetime cancer risks contributed from on-site Hookston Station sources plus off-site sources ranged from  $1.5 \text{ E-}07$  to  $3.9 \text{ E-}06$ . All of the calculated theoretical lifetime cancer risk were below or well within the range of risks considered acceptable by the USEPA.

For all 8 private wells sampled near the Hookston Station site, calculated exposures to VOCs in ground water resulting from use of ground water for irrigation and filling swimming pools were below  $1 \text{ E-}06$ , regardless of whether the risk allocated to on-site Hookston Station sources or all sources is considered.

Theoretical lifetime cancer risks resulting from volatilization of VOCs from surface water and inhalation by residents was calculated to be  $1.1 \text{ E-}06$  for risks allocated to on-site Hookston Station sources. The combined theoretical lifetime cancer risk from on-site and off-site sources was  $1.6 \text{ E-}06$ .

An evaluation of exposure and toxicological uncertainties indicated that most of the assumptions and methods used in the baseline RA will result in overestimation, rather than underestimation of risks.

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## **TABLES**

**Table 2.1.1**  
**On-Site Indoor Air Sampling Results**

<b>Indoor Air Sample Location</b>	<b>1,1- Dichloroethene ug/m<sup>3</sup></b>	<b>cis-1,2-Dichlororoethene ug/m<sup>3</sup></b>	<b>Trichloroethylene ug/m<sup>3</sup></b>
IA-1	<0.081	<0.16	1.9
IA-2	<0.074	1.7	4.9
IA-3	<0.075	<0.15	0.68
IA-5	<0.069	0.78	1.8
IA-6	<0.066	<0.13	2.4
AA-2	<0.065	<0.13	<0.18

**Table 2.1.2**  
**Chemicals Detected in 0 to 10 feet Depth Soils**  
**Hookston Station Site**

Chemical	Number of Detects/ Number of Analyses	Maximum Detected (mg/kg)	Average of Detects (mg/kg)	95% UCL on arithmetic mean (mg/kg)	Method used to calculate 95% UCL on arithmetic mean
<i>Volatile organic compounds</i>					
Acetone	1 / 86	<b>0.0629</b>	0.0629	NC	None
Benzene	1 / 92	<b>0.0010</b>	0.0010	NC	None
2-Butanone	7 / 86	<b>0.0316</b>	0.0179	NC	None
Carbon disulfide	3 / 86	<b>0.0029</b>	0.0018	NC	None
Chloroform	2 / 117	<b>0.0027</b>	0.0026	NC	None
1,1-Dichloroethane	0 / 117	ND	ND	ND	None
1,1-Dichloroethylene	0 / 117	ND	ND	ND	None
cis-1,2-Dichloroethylene	32 / 117	1.1000	0.0656	<b>0.082</b>	97.5% Chebyshev
trans-1,2-Dichloroethylene	8 / 117	<b>0.1190</b>	0.0231	NC	None
Dichloromethane	0 / 117	ND	ND	ND	None
Ethylbenzene	1 / 92	<b>0.0038</b>	0.0038	NC	None
(1)Freon 113	11 / 14	0.0240	0.0145	<b>0.015</b>	Student's t
2-Methyl-naphthalene	1 / 5	<b>0.128</b>	0.128	NC	None
Naphthalene	1 / 5	<b>0.162</b>	0.162	NC	None
Tetrachloroethylene	27 / 117	0.1470	0.0094	<b>0.009</b>	95% Chebyshev
Toluene	6 / 92	<b>0.0110</b>	0.0037	NC	None
1,1,1-Trichloroethane	1 / 117	<b>0.0090</b>	0.0090	NC	None
1,1,2-Trichloroethane	1 / 117	<b>0.0010</b>	0.0010	NC	None
Trichloroethylene	93 / 117	2.5800	0.1793	<b>0.433</b>	95% Chebyshev
Vinyl chloride	1 / 116	<b>0.0087</b>	0.0087	NC	None
Xylene (m-)	1 / 86	<b>0.0161</b>	0.0161	NC	None
Xylene (o-)	2 / 86	<b>0.0063</b>	0.0037	NC	None
Xylenes (total)	0 / 6	ND	ND	ND	None
<i>Semivolatile organic compounds</i>					
Acenaphthylene	1 / 5	<b>0.337</b>	0.337	NC	None
Anthracene	1 / 5	<b>0.213</b>	0.213	NC	None
*Benzo(a) pyrene	2 / 5	<b>0.743</b>	0.389	NC	None
*Benzo (b&k) fluoranthene	2 / 5	<b>1.04</b>	0.553	NC	None
*Chrysene	1 / 3	<b>0.705</b>	0.705	NC	None
*Indeno(1,2,3-c,d) pyrene	2 / 5	<b>0.563</b>	0.291	NC	None
*Benzo(a)pyrene equivalents	2 / 5	<b>0.910</b>	0.477	NC	None
Benzo(g,h,i) perylene	1 / 5	<b>0.885</b>	0.885	NC	None
Bis(2-ethylhexyl) phthalate	0 / 5	ND	ND	NC	None
Butylbenzyl phthalate	0 / 3	ND	ND	NC	None
Dibenzo(a,h) anthracene	1 / 5	<b>0.269</b>	0.269	NC	None
Di-n- butylphthalate	1 / 3	<b>0.141</b>	0.141	NC	None
Fluoranthene	2 / 5	<b>0.536</b>	0.304	NC	None
Fluorene	1 / 5	<b>0.0519</b>	0.0519	NC	None
Phenanthracene	3 / 5	<b>0.357</b>	0.165	NC	None
Pyrene	4 / 5	<b>1.17</b>	0.343	NC	None

**Table 2.1.2**  
**Chemicals Detected in 0 to 10 feet Depth Soils**  
**Hookston Station Site**

Chemical	Number of Detects/ Number of Analyses	Maximum Detected (mg/kg)	Average of Detects (mg/kg)	95% UCL on arithmetic mean (mg/kg)	Method used to calculate 95% UCL on arithmetic mean
<i>Petroleum hydrocarbons</i>					
Diesel fuel	5 / 20	985	259	<b>558</b>	99% Chebyshev
Gasoline	2 / 11	2.64	2.48	<b>2.27</b>	Student's t
Motor oil	8 / 20	8830	1340	<b>4903</b>	99% Chebyshev
<i>Metals</i>					
Antimony	5 / 19	5.16	2.02	<b>3.4</b>	95% Chebyshev
Arsenic	18 / 19	211	20.1	<b>132</b>	99% Chebyshev
Barium	19 / 19	172	85.4	<b>103</b>	Student's t
Beryllium	12 / 19	0.488	0.267	<b>0.3</b>	Approx. gamma
Cadmium	14 / 19	3.4	0.984	<b>1.2</b>	Approx. gamma
Chromium	19 / 19	130	36.1	<b>45.9</b>	Approx. gamma
Cobalt	18 / 19	29	13.2	<b>15.2</b>	Student's t
Copper	19 / 19	1100	127	<b>277</b>	95% Chebyshev
Lead	19 / 19	375	60.6	<b>104</b>	Approx. gamma
Mercury	13 / 19	1.4	0.327	<b>0.6</b>	95% Chebyshev
Molybdenum	10 / 19	6	1.51	<b>2.6</b>	95% Chebyshev
Nickel	19 / 19	62.9	31.5	<b>37.3</b>	Student's t
Selenium	7 / 19	0.44	0.335	<b>1.9</b>	95% Chebyshev
Silver	1 / 19	<b>0.805</b>	0.805	NC	NC
Thallium	3 / 19	<b>1.03</b>	0.461	NC	NC
Vanadium	19 / 19	330	64.4	<b>87.6</b>	Approx. gamma
Zinc	19 / 19	265	103	<b>132.3</b>	Student's t

Exposure point concentrations in **BOLD**

\*California relative potency factors are used to calculate benzo(a)pyrene equivalents for these compounds

Exposure and risk is calculated for "benzo(a)pyrene equivalents"

(1)1,1,2-Trichloro-1,2,2,-Trifluoroethane = Freon 113

NC - not calculated due to too few detected values

ND - not detected

**Table 2.1.3a**  
**Off-Site Indoor Air Sampling Information**

<b>Street</b>	<b>Date Sampled</b>	<b>Time Sampling Started - Ended</b>	<b>Indoor Temperature (degrees F)</b>	<b>Indoor Relative Humidity (%)</b>	<b>Outdoor Temperature (degrees F)</b>
1. Bermuda Dr	3/16/04	19:40 – 7:40	68	35	76
2. Bermuda Dr	3/16/04	19:55 – 7:50	74	33	72
3. Hampton Dr	1/21/04	--	--	--	--
4. Hampton Dr	2/19/04	19:55 – 7:54	70	52	57
5. Hampton Dr	2/19/04	20:10 – 8:00	70	50	57
6. Hampton Dr	2/25/04	19:35 – 7:32	70	50	62
7. Hookston Rd	2/19/04	19:40 – 7:40	64	46	63
8. Hookston Rd	1/20/04	--	--	--	--
9. Hookston Rd	3/3/04	19:30 – 7:30	68	46	58
10. Stimel Dr	2/17/04	20:10 – 8:04	74	63	--
11. Stimel Dr	2/17/04	19:55 – 7:55	70	59	--
12. Stimel Dr.	2/26/04	19:45 – 7:41	69	56	53
13. Stimel Dr	1/20/04	--	--	--	--
14. Thames Dr	2/19/04	20:25 – 8:21	65	51	55
15. Waterloo Ct	2/26/04	20:05 – 8:00	53	49	53
16. Waterloo Ct	3/16/04	19:20 – 7:25	75	26	74



**Table 2.1.3b**  
**VOCs in Indoor Air and Crawl Spaces of Residences**

Street	Living Space (ug/m <sup>3</sup> )			Crawl Space (ug/m <sup>3</sup> )		
	*TCE	*cis 1,2-DCE	*1,1-DCE	*TCE	*cis 1,2-DCE	*1,1-DCE
1. Bermuda Dr	<0.18	<0.14	<0.069	NA	NA	NA
2. Bermuda Dr	<0.18	<0.14	<0.070	NA	NA	NA
<sup>(a)</sup> 3. Hampton Dr	1.2	<0.14	<0.068	1.6	<0.13	<0.066
4. Hampton Dr	5	<0.14	0.11	2	<0.12	0.1
5. Hampton Dr	4.3	<0.14	0.13	2.2	0.2	0.11
6. Hampton Dr	0.43	<0.12	<0.059	NA	NA	NA
7. Hookston Rd	<0.18	<0.14	<0.068	NA	NA	NA
8. Hookston Rd	0.19	<0.13	0.064	NA	NA	NA
9. Hookston Rd	<0.18	<0.14	<0.068	NA	NA	NA
10. Stimel Dr	0.38	<0.13	<0.064	NA	NA	NA
11. Stimel Dr	3.3	<0.13	<0.065	5.1	0.38	<0.064
12. Stimel Dr.	2.0	<0.14	<0.069	0.53	<0.13	<0.065
13. Stimel Dr	3.8	<0.13	0.075	6.7	<0.13	0.082
<sup>(b)</sup> 14. Thames Dr	3.1	<0.12	<0.059	1.4	<0.12	0.059
15. Waterloo Ct	0.42	<0.14	<0.069	NA	NA	NA
16. Waterloo Ct	<0.18	<0.14	<0.068	NA	NA	NA

\*TCE = trichloroethylene; cis 1,2-DCE = cis 1,2-dichloroethylene; 1,1-DCE = 1,1-dichloroethylene  
NA = not analyzed

(a) An ambient outdoor sample collected at this location had no detectable 1,1-DCE (<0.068 ug/m<sup>3</sup>), cis-1,2-DCE (<0.14 ug/m<sup>3</sup>), and TCE (<0.18 ug/m<sup>3</sup>)

(b) An ambient outdoor sample collected at this location had no detectable 1,1-DCE (<0.62 ug/m<sup>3</sup>) or cis-1,2-DCE (<0.12 ug/m<sup>3</sup>). TCE was detected in ambient air at a concentration of 0.21 ug/m<sup>3</sup>

**Table 2.1.4a**  
**Information Concerning Private Wells Sampled Near the Hookston Station Site**

Residence	Well Diameter (inches)	Well Depth (ft bgs)	Well Material	Frequency of Use	Status
(a) Bermuda Drive	6"	55'	NA	None	Sampled from spigot 3/23/04.
(b) Bermuda Drive	6"	32'	Steel	None	Installed PDB 3/23/04. Sampled PDB 4/7/04.
(c) Stimel Drive	8"	60'	PVC	2/wk	Sampled from spigot 4/13/04.
(d) Stimel Drive	4"	75'	PVC	3/wk	Sampled from spigot 3/29/04.
(e) Stimel Drive	NA	NA	NA	Summer	Sampled from spigot 3/23/04.
(f) Gragg Lane	6"	126'	Steel	None	Installed PDB 3/23/04. Sampled PDB 4/7/04.
(g) Thames Drive	4"	30'	PVC	None	Inspected 3/29/04. Sampled from spigot 4/2/04.
(h) Waterloo Court	NA	35'	Unknown	1/wk	Sampled from spigot 3/24/04.

bgs - feet below ground surface

PDB - Passive diffusion bag

PVC - Polyvinyl chloride

NA - Not available

**Table 2.1.4b**  
**Private Well Ground Water Sampling Results**

Chemical	Private Well Sampling Results (all concentrations in ug/L)							
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Acetone	26 J, Q	2.2 J	12 J, Q	< 100	< 10	3.0 J	< 100	12 J, Q
Chloromethane	< 20	0.82 J	< 5	< 10	< 1	0.9 J	< 10	< 10
1,1-Dichloroethane	< 20	0.17 J	< 5	< 10	0.29 J	< 1	1.8 J	< 10
1,2-Dichloroethane	< 20	< 1	< 5	< 10	0.28 J	< 1	< 10	< 10
1,1-Dichloroethylene	11 J	1.4	5	4.9 J	1.7	< 1	9.2 J	4.4 J
cis-1,2-Dichloroethylene	24	0.26 J	17	37	12	< 1	13	6.5 J
trans-1,2-Dichloroethylene	< 20	< 1	2.4 J	< 10	0.49 J	< 1	< 10	< 10
Tetrachloroethylene	< 20	< 1	< 5	< 10	< 1	< 1	< 10	< 10
Trichloroethylene	670	29	130	210	8	< 1	500	380
Vinyl chloride	< 20	< 1	1.0 J	< 10	1.5	< 1	< 10	< 10

(a) Bermuda Drive; (b) Bermuda Drive; (c) Stimel Drive; (d) Stimel Drive; (e) Stimel Drive; (f) Gragg Lane; (g) Thames Drive; (h) Waterloo Court

"<" indicates chemical not detected at or above the specified reporting limit

"J" -estimated result; result is less than reporting limit.

"Q" - elevated reporting limit due to high analyte concentrations.

**Table 3.1**  
**Evaluation and Summary of Exposure Pathways**

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection/ Exclusion of Pathway
Current/Future	Soil	Soil	Site	Worker	Adult	Ingestion Dermal	On-Site	Quant	Workers currently on-site; future construction workers
				Visitor	Adult	Ingestion Dermal	On-Site	None	Pathway addressed by on-site worker (most exposed receptor)
		Outdoor Air	Site	Worker	Adult	Inhalation	On-Site	Quant	Workers currently on-site; future construction workers
				Visitor	Adult	Inhalation	On-Site	None	Pathway addressed by on-site worker (most exposed receptor)
		Indoor Air	Site	Worker	Adult	Inhalation of vapors migrating from subsurface soil	On-Site	Quant	Workers currently on-site
	Groundwater	Groundwater	Workplace tap	Worker	Adult	Ingestion Skin contact	On-Site	None	Ground water is not used as a potable supply
			Residential tap	Residents	Adult Child	Ingestion Skin contact	Off-Site	None	Off-site wells not used as potable water supply
			Swimming pools	Residents	Adult Child	Ingestion Skin contact	Off-Site	Quant	Residents may fill pools with well water
		Home-grown vegetables and fruits	Residence	Residents	Adult Child	Ingestion of produce grown in soils irrigated with groundwater	Off-Site	Qual	Residents are known to irrigate with ground water
		Outdoor Air	Swimming pools	Residents	Adult Child	Inhalation	Off-Site	Quant	Residents may fill pools with well water
		Air (indoor/ outdoor)	Irrigation Water	Residents	Adult Child	Inhalation	Off-Site	Quant	Off-yard residents may have private groundwater wells

**Table 3.1**  
**Evaluation and Summary of Exposure Pathways**

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection/ Exclusion of Pathway
Current/Future	Groundwater	Indoor Air	Workplace tap/shower	Worker	Adult	Inhalation	On-Site	None	There are no on-site wells
			Residential tap/shower	Residents	Adult Child	Inhalation	Off-Site	None	Off-site wells not used as potable water supply
			Site	Worker	Adult	Inhalation of vapors migrating from shallow ground water	On-Site	Quant	Buidlings are on-site
			Residential areas	Residents	Adult Child	Inhalation of vapors migrating from shallow ground water	Off-Site	Quant	Residences are located over affected ground water
			Nearby areas to Site	Worker	Adult	Inhalation of vapors migrating from shallow ground water	Off-Site	None	Pathway addressed by off- site resident (most exposed off-site receptor)
		Outdoor Air	Site	Worker	Adult	Inhalation of vapors migrating from shallow ground water	On-Site	None	Minor pathway of exposure due to rapid dilution in outdoor air
	Surface water	Fish	Walnut Creek	Residents	Adult Child	Ingestion	Off-Site	Qual	People have been observed fishing downstream in Walnut Creek
		Air (indoor/ outdoor)	Residences nearby to Walnut Creek	Residents	Adult Child	Inhalation	Off-Site	Quant	Volatile chemicals may be released as vapors into air

Quant-Pathway selected for risk quantification

Qual-Pathway selected for qualitative risk evaluation

**Table 3.2a**  
**Calculation of Intakes (mg/kg/day) of the Chemicals of Potential Concern**  
**On-site Industrial/Commercial Workers and On-site Construction Workers**

Exposure Pathway	Exposure Equation	Exposure variables
<b>Air</b>		
Inhalation of vapor-phase chemicals in air	$\frac{CA \times VR \times EF \times ED \times CF_{air}}{BW \times AT}$	<p>CA = Measured or modeled concentration of chemical in air (micrograms/m<sup>3</sup>)</p> <p>VR = Inhalation rate (m<sup>3</sup>/day)</p> <p>EF = Exposure frequency (days/year)</p> <p>ED = Exposure duration (years)</p> <p>CF<sub>air</sub> = Correction factor for air concentrations (0.001 mg/microgram)</p> <p>BW = Body weight (kg)</p> <p>AT =Averaging time (period over which exposure is averaged (AT<sub>nc</sub> for non-carcinogens = ED x 365; AT<sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)</p>
<b>Soil</b>		
Inhalation of vapor and particulate phase chemicals emitted from soil	$\frac{CS \times (1 / PEF \text{ or } 1 / VF) \times VR \times EF \times ED}{BW \times AT}$	<p>CS = Chemical concentration in soil (mg/kg)</p> <p>PEF = Particulate emission factor (m<sup>3</sup>/kg) for non-volatile chemicals or chemicals with low volatility (Henry's law constants less than 1 E-05 atm-m<sup>3</sup> /mole)</p> <p>VF = chemical-specific volatilization factor for volatile chemicals (see Appendix B for derivation; m<sup>3</sup>/kg)</p> <p>VR = Inhalation rate (m<sup>3</sup>/day)</p> <p>EF = Exposure frequency (days/year)</p> <p>ED = Exposure duration (years)</p> <p>BW = Body weight (kg)</p> <p>AT =Averaging time (period over which exposure is averaged (AT<sub>nc</sub> for non-carcinogens = ED x 365; AT<sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)</p>

**Table 3.2a** (continued)

Exposure Pathway	Exposure Equation	Exposure variables
<b>Soil</b>		
Ingestion of soil	$\frac{CS \times IR \times EF \times ED \times CF}{BW \times AT}$	<p>CS = Chemical concentration in soil (mg/kg)</p> <p>IR = Soil ingestion rate (mg/day)</p> <p>EF = Exposure frequency (days/year)</p> <p>ED = Exposure duration (years)</p> <p>CF= Conversion factor (<math>1 \times 10^{-6}</math> kg/mg)</p> <p>BW = Body weight (kg)</p> <p>AT =Averaging time (period over which exposure is averaged (AT<sub>nc</sub> for non-carcinogens = ED x 365;; AT<sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)</p>
Dermal absorption of chemicals in soil	$\frac{CS \times SA \times AF \times ABS \times EF \times ED \times CF}{BW \times AT}$	<p>CS = Chemical concentration in soil (mg/kg)</p> <p>SA = Skin surface area available for contact (cm<sup>2</sup>)</p> <p>AF = Adherence of soil to skin (mg/cm<sup>2</sup>)</p> <p>ABS = Fraction of chemical absorbed through the skin (unitless); dermal absorption fractions for the chemicals of concern were as follows: volatile organic chemicals, 0.10; PAHs, 0.15; semivolatile organic chemicals, 0.10; arsenic, 0.03; cadmium, 0.001; all other metals, 0.01</p> <p>EF = Exposure frequency (days/year)</p> <p>ED = Exposure duration (years)</p> <p>CF= Conversion factor (<math>1 \times 10^{-6}</math> kg/mg)</p> <p>BW = Body Weight (kg)</p> <p>AT =Averaging time (period over which exposure is averaged (AT<sub>nc</sub> for non-carcinogens = ED x 365;; AT<sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)</p>

**Table 3.2a**  
**Summary of Exposure On-Site Exposure Assumptions**

AF = Amount of soil adhering to skin (mg/cm<sup>2</sup>)  
 AT<sub>c</sub> = Averaging time for potential carcinogens (days)  
 AT<sub>nc</sub> = Averaging time for non-carcinogens (days)  
 BW = Body weight (kg)  
 ED = Number of years exposed (years)  
 EF = Exposure frequency (days/year)  
 IR = Soil ingestion rate (mg/day)  
 SA = Skin surface area exposed to soil (cm<sup>2</sup>)  
 VR = Inhalation rate (m<sup>3</sup>/day)

Exposure Parameter	On-site Commercial/ Industrial Worker	On-site Construction Worker
AF	0.2 <sup>c</sup>	0.51 <sup>d</sup>
AT <sub>c</sub>	25,550 <sup>a</sup>	25,550a
AT <sub>nc</sub>	9125	2555
BW	70 <sup>a</sup>	70 <sup>a</sup>
ED	25 <sup>a</sup>	7 <sup>d</sup>
EF	250 <sup>a</sup>	25 <sup>d</sup>
IR	50 <sup>a</sup>	330 <sup>b</sup>
PEF	1.32E+09	1.44E+06
SA	3300 <sup>c</sup>	5800 <sup>c</sup>
VF	Chemical-specific	Chemical-specific
VR	20 <sup>a</sup>	20 <sup>a</sup>

a USEPA, 1991

b USEPA, 2001a

c USEPA, 2001b

d SFRWQCB, 2003

na- not applicable



**Table 3.2b**  
**Calculation of Intakes (mg/kg/day) of the Chemicals of Potential Concern**  
**Off-site Residents**

<b>Exposure Pathway</b>	<b>Exposure Equation</b>	<b>Exposure variables</b>
<b>Air</b> Inhalation of vapor- phase chemicals in air	$\frac{CA \times VR \times EF \times ED \times CF_{air}}{BW \times AT}$	<p>CA = Measured or modeled concentration of chemical in air (micrograms/m<sup>3</sup>)</p> <p>VR = inhalation rate (m<sup>3</sup>/day);  (VR<sub>indoor</sub> = Inhalation rate for indoor air; VR<sub>sw</sub> = inhalation rate for chemicals assumed to volatilize from Walnut Creek)</p> <p>EF = Inhalation exposure frequency (days/year)</p> <p>ED = Exposure duration (years)</p> <p>CF<sub>air</sub> = Correction factor for air concentrations (0.001 mg/microgram)</p> <p>BW = Body weight (kg)</p> <p>AT =Averaging time (period over which exposure is averaged (AT<sub>nc</sub> for non-carcinogens = ED x 365;: AT<sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)</p>
Inhalation of volatile chemicals released during irrigation with ground water	$\frac{CA \times VR_{irrig} \times ET_{irrig} \times EF \times ED \times CF_{air}}{BW \times AT}$	<p>CA = Measured or modeled concentration of chemical in air (micrograms/m<sup>3</sup>)</p> <p>VR<sub>irrig</sub> = Inhalation rate for exposure to chemicals volatilizing from ground water used for irrigation (m<sup>3</sup>/hr)</p> <p>ET<sub>irrig</sub> = Hours of exposure to volatilizing chemicals per day (hours/day)</p> <p>EF = Inhalation exposure frequency (days/year)</p> <p>ED = Exposure duration (years)</p> <p>CF<sub>air</sub> = Correction factor for air concentrations (0.001 mg/microgram)</p> <p>BW = Body weight (kg)</p> <p>AT =Averaging time (period over which exposure is averaged (AT<sub>nc</sub> for non-carcinogens = ED x 365;: AT<sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)</p>

Table 3.2b

Exposure Pathway	Exposure Equation	Exposure variables
<b>Ground water</b> Skin contact with chemicals in ground water used for filling swimming pools	$\frac{DA_{event} \times SA \times EF \times ED}{BW \times AT}$	DA <sub>event</sub> = Dermally absorbed dose of chemical per swim (chemical specific- see Appendix C, mg/cm <sup>2</sup> ) SA = Skin surface area available for contact (cm <sup>2</sup> ) EF = Frequency of swimming (days/year) ED = Exposure duration (years) BW = Body weight (kg) AT = Averaging time (period over which exposure is averaged (AT <sub>nc</sub> for non-carcinogens = ED x 365; AT <sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)
Incidental ingestion of chemicals in ground water used for filling swimming pools	$\frac{CW \times IR_{pool} \times ET_{pool} \times LF \times EF \times ED \times CF_{water}}{BW \times AT}$	CW = Concentration of chemical in ground water (micrograms per L) IR <sub>pool</sub> = Incidental ingestion rate of pool water (L/hr) ET <sub>pool</sub> = Duration of swimming exposure (hr/day) LF = loss factor; factor accounting for loss of volatile compounds from pool water over swimming season (see Appendix C for derivation) EF = Frequency of swimming (days/year) ED = Exposure duration (years) CF <sub>water</sub> = Conversion factor (0.001 mg/microgram) BW = Body weight (kg) AT = Averaging time (period over which exposure is averaged (AT <sub>nc</sub> for non-carcinogens = ED x 365; AT <sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)
Inhalation of chemicals volatilizing from ground water used for filling swimming pools	$\frac{CW \times VF_{pool} \times VR_{pool} \times ET \times EF \times ED \times CF_{water}}{BW \times AT}$	CW = Chemical concentration in pool water (micrograms/L) VF <sub>pool</sub> = Volatilization factor derived for chemicals volatilizing from pool water (chemical specific; see Appendix) VR <sub>pool</sub> = Inhalation rate during swimming (m <sup>3</sup> /hr) ET <sub>pool</sub> = Duration of swimming exposure (hr/day) EF = Frequency of swimming (days/year) ED = Exposure Duration (years) CF <sub>water</sub> = Conversion factor (0.001 mg/microgram) BW = Body Weight (kg) AT = Averaging time (period over which exposure is averaged (AT <sub>nc</sub> for non-carcinogens = ED x 365; AT <sub>c</sub> for carcinogens: 70 years x 365 days/year = 25,550 days)

**Table 3.2b**  
**Summary of Exposure Off-Site Exposure Assumptions**

AF = Amount of soil adhering to skin (mg/cm<sup>2</sup>)  
 AT<sub>c</sub> = Averaging time for potential carcinogens (days)  
 AT<sub>nc</sub> = Averaging time for non-carcinogens (days)  
 BW = Body weight (kg)  
 ED = Number of years exposed (years)  
 EF = Exposure frequency (days/year)  
 ET<sub>irrig</sub> = Exposure time for irrigation water (hr/day)  
 ET<sub>pool</sub> = Swimming exposure time (hr./day)  
 IR<sub>pool</sub> = Swimming pool water ingestion rate (L/hr)  
 LF = Loss factor for chemicals volatilizing from swimming pool water

SA = skin surface area exposed to swimming pool water (cm<sup>2</sup>)  
 VF<sub>irrig</sub> = Volatilization factor for chemicals from irrigation water (L/m<sup>3</sup>)  
 VF<sub>pool</sub> = Volatilization factor for chemicals from pool water (L/m<sup>3</sup>)  
 VR<sub>indoor</sub> = Inhalation rate for indoor air (m<sup>3</sup>/day)  
 VR<sub>irrig</sub> = Inhalation rate for exposure to chemicals released to air from irrigation water (m<sup>3</sup>/day)  
 VR<sub>pool</sub> = Inhalation rate for exposure to chemicals released to air from pool water (m<sup>3</sup>/hr)  
 VR<sub>sw</sub> = Inhalation rate for exposure to chemicals released to air from Walnut Creek (m<sup>3</sup>/day)

Exposure Parameter	Indoor Air Exposure Scenario		Ground Water Irrigation Scenario		Swimming Pool Exposure Scenario	Inhalation Exposure to Volatile Compounds Released from Walnut Creek	
	Child Resident	Adult Resident	Child Resident	Adult Resident	Child Resident	Child Resident	Adult Resident
AT <sub>c</sub>	25,550 <sup>a</sup>	25,550 <sup>a</sup>	25,550 <sup>a</sup>	25,550 <sup>a</sup>	25,550 <sup>a</sup>	25,550 <sup>a</sup>	25,550 <sup>a</sup>
AT <sub>nc</sub>	2190 <sup>a</sup>	na	2190 <sup>a</sup>	na	4745 <sup>b</sup>	2190	na
BW	70 <sup>a</sup>	70 <sup>a</sup>	15 <sup>a</sup>	70 <sup>a</sup>	41.5	15 <sup>a</sup>	70 <sup>a</sup>
ED	6 <sup>a</sup>	24 <sup>a</sup>	6 <sup>a</sup>	24 <sup>a</sup>	13 <sup>b</sup>	6 <sup>a</sup>	24 <sup>a</sup>
EF	350 <sup>a</sup>	350 <sup>a</sup>	63 <sup>b</sup>	63 <sup>b</sup>	108 <sup>b</sup>	350 <sup>b</sup>	350 <sup>b</sup>
ET <sub>irrig</sub>	na	na	8 <sup>b</sup>	8 <sup>b</sup>	na	na	na
ET <sub>pool</sub>	na	na	na	na	1 <sup>c</sup>	na	na
IR <sub>pool</sub>	na	na	na	na	0.05 <sup>d</sup>	na	na
LF	na	na	na	na	0.12	na	na
SA	na	na	na	na	15,500 <sup>b</sup>	na	na
VF <sub>irrig</sub>	na	na	0.00844 <sup>b</sup>	0.00844 <sup>b</sup>	na	na	na
VF <sub>pool</sub>	na	na	na	na	0.000977 <sup>b</sup>	na	na
VR <sub>indoor</sub>	8.7 <sup>c</sup>	13.3 <sup>c</sup>	na	na	na	na	na
VR <sub>irrig</sub>	na	na	0.415 <sup>b</sup>	0.83 <sup>b</sup>	na	na	na
VR <sub>pool</sub>	na	na	na	na	1.9 <sup>b</sup>	na	na
VR <sub>sw</sub>	na	na	na	na	na	10 <sup>e</sup>	20 <sup>a</sup>

a-USEPA, 1991; b-see Appendix C for derivation of exposure variable; c-USEPA, 1997a; d-USEPA, 1989; e-SFRWQCB, 2003

na- not applicable

**Table 3.3**  
**Inhalation of Volatile Organic Compounds in On-Site Indoor Air**  
**On-Site Commercial/Industrial Worker**

On-site Indoor Air Sampling Location IA-1

Chemical	Air Concentration (ug/m <sup>3</sup> )	Commercial/Industrial Worker	
		ADI	LADI
cis-1,2-Dichloroethylene	ND	ND	NC
Trichloroethylene	1.900	3.72E-04	1.33E-04

On-site Indoor Air Sampling Location IA-2

Chemical	Air Concentration (ug/m <sup>3</sup> )	Commercial/Industrial Worker	
		ADI	LADI
cis-1,2-Dichloroethylene	1.700	3.33E-04	1.19E-04
Trichloroethylene	4.900	9.59E-04	3.42E-04

On-site Indoor Air Sampling Location IA-3

Chemical	Air Concentration (ug/m <sup>3</sup> )	Commercial/Industrial Worker	
		ADI	LADI
cis-1,2-Dichloroethylene	ND	ND	NC
Trichloroethylene	0.680	1.33E-04	4.75E-05

On-site Indoor Air Sampling Location IA-5

Chemical	Air Concentration (ug/m <sup>3</sup> )	Commercial/Industrial Worker	
		ADI	LADI
cis-1,2-Dichloroethylene	0.780	1.53E-04	5.45E-05
Trichloroethylene	1.800	3.52E-04	1.26E-04

On-site Indoor Air Sampling Location IA-6

Chemical	Air Concentration (ug/m <sup>3</sup> )	Commercial/Industrial Worker	
		ADI	LADI
cis-1,2-Dichloroethylene	ND	ND	NC
Trichloroethylene	2.400	4.70E-04	1.68E-04

ADI = Average daily intake in mg/kg/day

LADI = Lifetime average daily intake in mg/kg/day

ND = not detected

NC = not carcinogenic

**Table 3.4**  
**Soil Exposure Calculations**  
**On-Site Commercial/Industrial Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
<i>Volatile Organic Compounds</i>				
Acetone	0.063	3.08E-08	4.06E-08	1.61E-06
Benzene	0.001	4.81E-10	6.35E-10	1.15E-07
2-Butanone	0.032	1.55E-08	2.04E-08	5.21E-07
Carbon disulfide	0.003	1.39E-09	1.84E-09	7.68E-07
Chloroform	0.003	1.32E-09	1.74E-09	3.25E-07
cis-1,2-Dichloroethylene	0.082	4.01E-08	5.30E-08	9.05E-06
trans-1,2-Dichloroethylene	0.119	5.82E-08	7.68E-08	1.64E-05
Ethylbenzene	0.004	1.84E-09	2.43E-09	2.24E-07
Freon 113	0.015	7.34E-09	9.69E-09	3.06E-06
2-Methylnaphthalene	0.128	6.26E-08	8.27E-08	9.43E-07
Naphthalene	0.162	7.93E-08	1.05E-07	1.20E-06
Tetrachloroethylene	0.009	4.40E-09	5.81E-09	1.13E-06
Toluene	0.011	5.38E-09	7.10E-09	8.87E-07
1,1,1-Trichloroethane	0.009	4.40E-09	5.81E-09	1.31E-06
1,1,2-Trichloroethane	0.001	4.89E-10	6.46E-10	4.94E-08
Trichloroethylene	0.433	2.12E-07	2.80E-07	4.26E-05
Vinyl chloride	0.009	4.26E-09	5.62E-09	2.69E-06
m-Xylene	0.009	4.26E-09	5.62E-09	4.58E-07
o-Xylene	0.016	7.88E-09	1.04E-08	8.47E-07
<i>Semivolatile Organic Compounds</i>				
Acenaphthylene	0.337	1.65E-07	3.26E-07	2.54E-06
Anthracene	0.213	1.04E-07	2.06E-07	9.80E-08
Benzo(a)pyrene equivalents	0.910	4.45E-07	8.82E-07	1.35E-10
Benzo(g,h,i)perylene	0.885	4.33E-07	5.72E-07	1.31E-10
Dibenz(a,h)anthracene	0.269	1.32E-07	2.61E-07	3.99E-11
Di-n-butylphthalate	0.141	6.90E-08	9.11E-08	2.09E-11
Fluoranthene	0.536	2.62E-07	5.19E-07	7.95E-11
Fluorene	0.052	2.54E-08	5.03E-08	3.58E-08
Phenanthrene	0.357	1.75E-07	3.46E-07	2.26E-07
Pyrene	1.170	5.72E-07	1.13E-06	1.74E-10
<i>Petroleum Hydrocarbons</i>				
Diesel fuel	558	2.73E-04	3.60E-04	8.28E-08
Gasoline	2.27	1.11E-06	1.47E-06	3.42E-05
Motor oil	4903	2.40E-03	3.17E-03	7.27E-07

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
1.72E-10	2.27E-10	4.12E-08
NC	NC	NC
NC	NC	NC
4.72E-10	6.23E-10	1.16E-07
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	4.29E-07
1.57E-09	2.08E-09	4.04E-07
NC	NC	NC
NC	NC	NC
1.75E-10	2.31E-10	1.77E-08
7.57E-08	9.99E-08	1.52E-05
1.52E-09	2.01E-09	9.62E-07
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
1.59E-07	3.15E-07	4.82E-11
NC	NC	NC
4.70E-08	9.31E-08	1.43E-11
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC

**Table 3.4**  
**Soil Exposure Calculations**  
**On-Site Commercial/Industrial Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
<i>Metals</i>				
Antimony	3.40	1.66E-06	2.20E-07	5.04E-10
Arsenic	132	6.46E-05	2.56E-05	1.96E-08
Barium	103	5.04E-05	6.65E-06	1.53E-08
Beryllium	0.300	1.47E-07	1.94E-08	4.45E-11
Cadmium	1.20	5.87E-07	7.75E-09	1.78E-10
Chromium	45.90	2.25E-05	2.96E-06	6.81E-09
Cobalt	15.2	7.44E-06	9.82E-07	2.25E-09
Copper	277	1.36E-04	1.79E-05	4.11E-08
Lead	104	5.09E-05	6.72E-06	1.54E-08
Mercury	0.600	2.94E-07	3.87E-08	8.90E-11
Molybdenum	2.60	1.27E-06	1.68E-07	3.86E-10
Nickel	37.3	1.82E-05	2.41E-06	5.53E-09
Selenium	1.90	9.30E-07	1.23E-07	2.82E-10
Silver	0.8	3.94E-07	5.20E-08	1.19E-10
Thallium	1.0	5.04E-07	6.65E-08	1.53E-10
Vanadium	88	4.29E-05	5.66E-06	1.30E-08
Zinc	132.30	6.47E-05	8.54E-06	1.96E-08

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
2.31E-05	9.13E-06	6.99E-09
NC	NC	NC
NC	NC	1.59E-11
2.10E-07	2.77E-09	6.36E-11
NC	NC	NC
NC	NC	NC
NC	NC	NC
1.82E-05	2.40E-06	5.51E-09
NC	NC	NC
NC	NC	NC
NC	NC	1.98E-09
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC

NC = not carcinogenic

**Table 3.5**  
**Soil Exposure Calculations**  
**On-Site Construction Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
<i>Volatile Organic Compounds</i>				
Acetone	0.063	1.62E-08	1.46E-08	2.43E-07
Benzene	0.001	2.54E-10	2.28E-10	1.75E-08
2-Butanone	0.032	8.16E-09	7.32E-09	7.88E-08
Carbon disulfide	0.003	7.36E-10	6.60E-10	1.16E-07
Chloroform	0.003	6.97E-10	6.25E-10	4.92E-08
cis-1,2-Dichloroethylene	0.082	2.12E-08	1.90E-08	1.37E-06
trans-1,2-Dichloroethylene	0.119	3.07E-08	2.76E-08	2.49E-06
Ethylbenzene	0.004	9.74E-10	8.73E-10	3.39E-08
Freon 113	0.015	3.87E-09	3.47E-09	4.63E-07
2-Methylnaphthalene	0.128	3.31E-08	2.96E-08	1.43E-07
Naphthalene	0.162	4.18E-08	3.75E-08	1.82E-07
Tetrachloroethylene	0.009	2.32E-09	2.08E-09	1.71E-07
Toluene	0.011	2.84E-09	2.55E-09	1.34E-07
1,1,1-Trichloroethane	0.009	2.32E-09	2.08E-09	1.97E-07
1,1,2-Trichloroethane	0.001	2.58E-10	2.32E-10	7.47E-09
Trichloroethylene	0.433	1.12E-07	1.00E-07	6.44E-06
Vinyl chloride	0.009	2.25E-09	2.01E-09	4.07E-07
m-Xylene	0.009	2.25E-09	2.01E-09	6.92E-08
o-Xylene	0.016	4.16E-09	3.73E-09	1.28E-07
<i>Semivolatile Organic Compounds</i>				
Acenaphthylene	0.34	8.71E-08	1.17E-07	3.83E-07
Anthracene	0.21	5.50E-08	7.40E-08	1.48E-08
Benzo(a)pyrene equivalents	0.91	2.35E-07	3.16E-07	9.89E-09
Benzo(g,h,i)perylene	0.89	2.29E-07	2.05E-07	9.62E-09
Dibenz(a,h)anthracene	0.27	6.95E-08	9.34E-08	2.92E-09
Di-n-butylphthalate	0.14	3.64E-08	3.26E-08	1.53E-09
Fluoranthene	0.54	1.38E-07	1.86E-07	5.82E-09
Fluorene	0.05	1.34E-08	1.80E-08	5.41E-09
Phenanthrene	0.36	9.22E-08	1.24E-07	3.42E-08
Pyrene	1.17	3.02E-07	4.06E-07	1.27E-08
<i>Petroleum Hydrocarbons</i>				
Diesel fuel	558	1.44E-04	1.29E-04	6.06E-06
Gasoline	2.27	5.86E-07	5.26E-07	5.17E-06
Motor oil	4903	1.27E-03	1.14E-03	5.33E-05

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
2.54E-11	2.28E-11	1.75E-09
NC	NC	NC
NC	NC	NC
6.97E-11	6.25E-11	4.92E-09
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	1.82E-08
2.32E-10	2.08E-10	1.71E-08
NC	NC	NC
NC	NC	NC
2.58E-11	2.32E-11	7.47E-10
1.12E-08	1.00E-08	6.44E-07
2.25E-10	2.01E-10	4.07E-08
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
2.35E-08	3.16E-08	9.89E-10
NC	NC	NC
6.95E-09	9.34E-09	2.92E-10
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC

**Table 3.5**  
**Soil Exposure Calculations**  
**On-Site Construction Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
<i>Metals</i>				
Antimony	3.40	8.78E-07	7.87E-08	3.69E-08
Arsenic	132	3.41E-05	9.17E-06	1.43E-06
Barium	103	2.66E-05	2.38E-06	1.12E-06
Beryllium	0.300	7.75E-08	6.95E-09	3.26E-09
Cadmium	1.20	3.10E-07	2.78E-09	1.30E-08
Chromium	45.90	1.19E-05	1.06E-06	4.99E-07
Cobalt	15.2	3.93E-06	3.52E-07	1.65E-07
Copper	277	7.16E-05	6.41E-06	3.01E-06
Lead	104	2.69E-05	2.41E-06	1.13E-06
Mercury	0.600	1.55E-07	1.39E-08	6.52E-09
Molybdenum	2.60	6.72E-07	6.02E-08	2.82E-08
Nickel	37.3	9.64E-06	8.64E-07	4.05E-07
Selenium	1.90	4.91E-07	4.40E-08	2.06E-08
Silver	0.8	2.08E-07	1.86E-08	8.75E-09
Thallium	1.0	2.66E-07	2.38E-08	1.12E-08
Vanadium	88	2.26E-05	2.03E-06	9.52E-07
Zinc	132.30	3.42E-05	3.06E-06	1.44E-06

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
3.41E-06	9.17E-07	1.43E-07
NC	NC	NC
NC	NC	3.26E-10
3.10E-08	2.78E-10	1.30E-09
NC	NC	NC
NC	NC	NC
NC	NC	NC
2.69E-06	2.41E-07	1.13E-07
NC	NC	NC
NC	NC	NC
NC	NC	4.05E-08
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC

NC = not carcinogenic



**Table 3.6**  
**Inhalation of Volatile Organic Compounds in Indoor Residential Air**  
**Off-Site Residents**

3. Hampton Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	1.200	6.67E-04	5.72E-05	2.19E-04	7.50E-05

4. Hampton Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	0.110	6.12E-05	NC	2.00E-05	NC
Trichloroethylene	5.000	2.78E-03	2.38E-04	9.11E-04	3.12E-04

5. Hampton Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	0.130	7.23E-05	NC	2.37E-05	NC
Trichloroethylene	4.300	2.39E-03	2.05E-04	7.83E-04	2.69E-04

6 Hampton Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	0.430	2.39E-04	2.05E-05	7.83E-05	2.69E-05

8 Hookston Road

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	0.064	3.56E-05	NC	1.17E-05	NC
Trichloroethylene	0.190	1.06E-04	9.06E-06	3.46E-05	1.19E-05

10 Stimel Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	0.380	2.11E-04	1.81E-05	6.92E-05	2.37E-05

**Table 3.6**  
**Inhalation of Volatile Organic Compounds in Indoor Residential Air**  
**Off-Site Residents**

11 Stimel Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	3.300	1.84E-03	1.57E-04	6.01E-04	2.06E-04

12 Stimel Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	2.000	1.11E-03	9.53E-05	3.64E-04	1.25E-04

13 Stimel Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	0.075	4.17E-05	NC	1.37E-05	NC
Trichloroethylene	3.800	2.11E-03	1.81E-04	6.92E-04	2.37E-04

14 Thames Drive

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	3.100	1.72E-03	1.48E-04	5.65E-04	1.94E-04

15 Waterloo Court

Chemical	Air Concentration (ug/m <sup>3</sup> )	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
1,1-Dichloroethylene	ND	ND	ND	ND	ND
Trichloroethylene	0.420	2.34E-04	2.00E-05	7.65E-05	2.62E-05

ADI = Average daily intake in mg/kg/day

LADI = Lifetime average daily intake in mg/kg/day

ND = not detected

NC = not carcinogenic

**Table 3.7**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(a) Bermuda

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	26.0	8.43E-06	NC	3.61E-06	NC
Chloromethane	ND	ND	NC	ND	NC
1,1-Dichloroethane	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	11.0	3.57E-06	NC	1.53E-06	NC
cis-1,2-Dichloroethene	24.0	7.78E-06	NC	3.33E-06	NC
trans-1,2-Dichloroethene	ND	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	670.0	2.17E-04	1.86E-05	9.31E-05	3.19E-05
Vinyl chloride	ND	ND	ND	ND	ND

(b) Bermuda

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	2.2	7.13E-07	NC	3.06E-07	NC
Chloromethane	0.8	2.66E-07	NC	1.14E-07	NC
1,1-Dichloroethane	0.2	5.51E-08	4.72E-09	2.36E-08	8.10E-09
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	1.4	4.54E-07	NC	1.95E-07	NC
cis-1,2-Dichloroethene	0.3	8.43E-08	NC	3.61E-08	NC
trans-1,2-Dichloroethene	ND	ND	NC	ND	NC
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	29.0	9.40E-06	8.06E-07	4.03E-06	1.38E-06
Vinyl chloride	ND	ND	ND	ND	ND

(c) Stimel

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	12.0	3.89E-06	NC	1.67E-06	NC
Chloromethane	ND	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	5.0	1.62E-06	1.39E-07	6.95E-07	2.38E-07
cis-1,2-Dichloroethene	17.0	5.51E-06	NC	2.36E-06	NC
trans-1,2-Dichloroethene	2.4	7.78E-07	NC	3.33E-07	NC
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	130.0	4.21E-05	3.61E-06	1.81E-05	6.19E-06
Vinyl chloride	1.0	3.24E-07	2.78E-08	1.39E-07	4.76E-08

**Table 3.7**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(d) Stimel

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	ND	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	4.9	1.59E-06	NC	6.81E-07	NC
cis-1,2-Dichloroethene	37.0	1.20E-05	NC	5.14E-06	NC
trans-1,2-Dichloroethene	ND	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	210.0	6.81E-05	5.83E-06	2.92E-05	1.00E-05
Vinyl chloride	ND	ND	ND	ND	ND

(e) Stimel

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	ND	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND	ND
1,1-Dichloroethane	0.3	9.40E-08	8.06E-09	4.03E-08	1.38E-08
1,2-Dichloroethane	0.3	9.08E-08	7.78E-09	3.89E-08	1.33E-08
1,1-Dichloroethene	1.7	5.51E-07	NC	2.36E-07	NC
cis-1,2-Dichloroethene	12.0	3.89E-06	NC	1.67E-06	NC
trans-1,2-Dichloroethene	0.5	1.59E-07	NC	6.81E-08	NC
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	8.0	2.59E-06	2.22E-07	1.11E-06	3.81E-07
Vinyl chloride	1.5	4.86E-07	4.17E-08	2.08E-07	7.14E-08

(f) Gragg Lane

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	3.0	9.72E-07	NC	4.17E-07	NC
Chloromethane	0.9	2.92E-07	NC	1.25E-07	NC
1,1-Dichloroethane	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	ND	ND	ND	ND	ND
cis-1,2-Dichloroethene	ND	ND	ND	ND	ND
trans-1,2-Dichloroethene	ND	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	ND	ND	ND	ND	ND
Vinyl chloride	ND	ND	ND	ND	ND

**Table 3.7**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(g) Thames Drive

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	ND	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND	ND
1,1-Dichloroethane	1.8	5.83E-07	5.00E-08	2.50E-07	8.57E-08
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	9.2	2.98E-06	NC	1.28E-06	NC
cis-1,2-Dichloroethene	13.0	4.21E-06	NC	1.81E-06	NC
trans-1,2-Dichloroethene	ND	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	500.0	1.62E-04	1.39E-05	6.95E-05	2.38E-05
Vinyl chloride	ND	ND	ND	ND	ND

(h) Waterloo

Chemical	Ground water concentration ug/L	Child Resident		Adult Resident	
		ADI	LADI	ADI	LADI
Acetone	12.0	3.89E-06	NC	1.67E-06	NC
Chloromethane	ND	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	ND
1,1-Dichloroethene	4.4	1.43E-06	NC	6.11E-07	NC
cis-1,2-Dichloroethene	6.5	2.11E-06	NC	9.03E-07	NC
trans-1,2-Dichloroethene	ND	ND	NC	ND	NC
Tetrachloroethene	ND	ND	ND	ND	ND
Trichloroethene	380.0	1.23E-04	1.06E-05	5.28E-05	1.81E-05
Vinyl chloride	ND	ND	ND	ND	ND

ADI = Average daily intake in mg/kg/day

LADI = Lifetime average daily intake in mg/kg/day

ND = not detected

NC = not carcinogenic

**Table 3.8**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

(a) Bermuda

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	26.0	1.13E-06	2.00E-07	3.50E-07
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	11.0	4.78E-07	3.10E-06	1.48E-07
cis-1,2-Dichloroethene	24.0	1.04E-06	4.34E-06	3.23E-07
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	670.0	2.91E-05	2.28E-04	9.03E-06
Vinyl chloride	ND	ND	ND	ND

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
5.41E-06	4.24E-05	1.68E-06
ND	ND	ND

(b) Bermuda

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	2.2	9.57E-08	1.69E-08	2.97E-08
Chloromethane	0.8	3.57E-08	5.11E-08	1.11E-08
1,1-Dichloroethane	0.2	7.39E-09	2.70E-08	2.29E-09
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	1.4	6.09E-08	3.94E-07	1.89E-08
cis-1,2-Dichloroethene	0.3	1.13E-08	4.70E-08	3.50E-09
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	29.0	1.26E-06	9.88E-06	3.91E-07
Vinyl chloride	ND	ND	ND	ND

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
1.37E-09	5.02E-09	4.26E-10
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
2.34E-07	1.84E-06	7.26E-08
ND	ND	ND

(c) Stimel

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	12.0	5.22E-07	9.21E-08	1.62E-07
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	5.0	2.18E-07	1.41E-06	6.74E-08
cis-1,2-Dichloroethene	17.0	7.39E-07	3.07E-06	2.29E-07
trans-1,2-Dichloroethene	2.4	1.04E-07	4.34E-07	3.23E-08
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	130.0	5.65E-06	4.43E-05	1.75E-06
Vinyl chloride	1.0	4.35E-08	1.12E-07	1.35E-08

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
1.05E-06	8.22E-06	3.25E-07
8.08E-09	2.08E-08	2.50E-09

**Table 3.8**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

(d) Stimmel

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	4.9	2.13E-07	1.38E-06	6.60E-08
cis-1,2-Dichloroethene	37.0	1.61E-06	6.68E-06	4.99E-07
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	210.0	9.13E-06	7.15E-05	2.83E-06
Vinyl chloride	ND	ND	ND	ND

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
1.70E-06	1.33E-05	5.26E-07
ND	ND	ND

(e) Stimmel

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	0.3	1.26E-08	4.61E-08	3.91E-09
1,2-Dichloroethane	0.3	1.22E-08	2.79E-08	3.77E-09
1,1-Dichloroethene	1.7	7.39E-08	4.79E-07	2.29E-08
cis-1,2-Dichloroethene	12.0	5.22E-07	2.17E-06	1.62E-07
trans-1,2-Dichloroethene	0.5	2.13E-08	8.85E-08	6.60E-09
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	8.0	3.48E-07	2.73E-06	1.08E-07
Vinyl chloride	1.5	6.52E-08	1.68E-07	2.02E-08

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
2.34E-09	8.56E-09	7.26E-10
2.26E-09	5.18E-09	7.01E-10
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
6.46E-08	5.06E-07	2.00E-08
1.21E-08	3.11E-08	3.75E-09

(f) Gragg Lane

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	3.0	1.31E-07	2.30E-08	4.04E-08
Chloromethane	0.9	3.91E-08	5.61E-08	1.21E-08
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	ND	ND	ND	ND
cis-1,2-Dichloroethene	ND	ND	ND	ND
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	ND	ND	ND	ND
Vinyl chloride	ND	ND	ND	ND

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
ND	ND	ND

**Table 3.8**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

(g) Thames Drive

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	1.8	7.83E-08	2.86E-07	2.43E-08
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	9.2	4.00E-07	2.59E-06	1.24E-07
cis-1,2-Dichloroethene	13.0	5.65E-07	2.35E-06	1.75E-07
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	500.0	2.18E-05	1.70E-04	6.74E-06
Vinyl chloride	ND	ND	ND	ND

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
1.45E-08	5.32E-08	4.51E-09
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
4.04E-06	3.16E-05	1.25E-06
ND	ND	ND

(h) Waterloo

Chemical	Ground water concentration ug/L	Average Daily Intake (mg/kg/day)		
		Ingestion	Dermal contact	Inhalation
Acetone	12.0	5.22E-07	9.21E-08	1.62E-07
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	4.4	1.91E-07	1.24E-06	5.93E-08
cis-1,2-Dichloroethene	6.5	2.83E-07	1.17E-06	8.76E-08
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	380.0	1.65E-05	1.29E-04	5.12E-06
Vinyl chloride	ND	ND	ND	ND

Lifetime Average Daily Intake (mg/kg/day)		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
3.07E-06	2.40E-05	9.51E-07
ND	ND	ND

ND = not detected  
NC = not carcinogenic



**Table 3.9**  
**Inhalation of Volatile Organic Compounds Volatilizing from Walnut Creek Surface Water**  
**Off-Site Residents**

Chemical	Surface Water Concentration(ug/L)	Child Resident		Adult Resident	
		Average Daily Intake (mg/kg/day)	Lifetime Average Daily Intake (mg/kg/day)	Average Daily Intake (mg/kg/day)	Lifetime Average Daily Intake (mg/kg/day)
cis-1,2-Dichloroethylene	1.4	1.42E-04	NC	6.07E-05	NC
Tetrachloroethylene	2.6	2.22E-04	1.91E-05	9.53E-05	3.27E-05
Trichloroethylene	3.3	3.10E-04	2.66E-05	1.33E-04	4.56E-05

NC = not carcinogenic

**Table 4.1**  
**Inhalation Reference Doses and Slope Factors**

Chemical	Inhalation Reference Dose (mg/kg/day)	Uncertainty Factor	Non-carcinogenic Effects	Notes for RfD	Slope Factor (mg/kg/day) <sup>-1</sup>	EPA Group	Carcinogenic Effects	Notes for slope factor
<b>Volatile organic chemicals</b>								
Acetone	9.00E-01	-	-	EPA Region 9	-	-	-	-
Benzene	8.60E-03	300	Decreased lymphocyte count	IRIS	1.00E-01	A	Leukemia	Source: Cal EPA
2-Butanone (Methyl ethyl ketone)	1.43E+00	-	-	EPA Region 9	-	-	-	-
Carbon disulfide	2.00E-01	30	Peripheral nervous system	IRIS	-	-	-	-
Chloroform	1.40E-02	-	-	EPA Region 9	1.90E-02	-	-	Source: Cal EPA
Chloromethane	2.60E-02	1000	Central nervous system	IRIS	-	-	-	-
1,1-Dichloroethane	1.40E-01	-	-	EPA Region 9	5.70E-03	na	na	Source: Cal EPA
1,2-Dichloroethane	1.40E-03	-	-	EPA Region 9	7.20E-02	na	na	Source: Cal EPA
1,1-Dichloroethylene	5.70E-02	30	Respiratory system	IRIS	na	na	na	
cis-1,2-Dichloroethylene	1.00E-02	-	-	EPA Region 9	-	-	-	-
trans-1,2-Dichloroethylene	2.00E-02	-	-	EPA Region 9	-	-	-	-
Ethylbenzene	2.90E-01	300	Developmental	IRIS	-	-	-	-
Freon 113	8.60E+00	-	-	HEAST	-	-	-	-
2-Methylnaphthalene	4.00E-03	-	-	Oral RfD	-	-	-	-
Naphthalene	8.60E-04	3000	Nasal effects	IRIS	-	-	-	OEHHA
Tetrachloroethylene	1.00E-02	-	-	EPA Region 9	2.10E-02	na	na	Source: Cal EPA
Toluene	1.10E-01	300	CNS effects; eyes	IRIS	-	-	-	-
1,1,1-Trichloroethane	6.30E-01	-	-	EPA Region 9	-	-	-	-
1,1,2-Trichloroethane	4.00E-03	-	-	EPA Region 9	5.70E-02	C	na	Source: Cal EPA
Trichloroethylene	1.00E-02	-	-	EPA Region 9	7.00E-03	na	na	Source: Cal EPA
Vinyl Chloride	2.90E-02	30	Liver	IRIS	2.70E-01	A	Liver angiosarcomas	Source: Cal EPA
m-Xylene	2.90E-02	300	motor coordination	IRIS	-	-	-	-
o-Xylene	2.90E-02	300	motor coordination	IRIS	-	-	-	-
Xylenes	2.90E-02	300	motor coordination	IRIS	-	-	-	-

**Table 4.1**  
**Inhalation Reference Doses and Slope Factors**

Chemical	Inhalation Reference Dose (mg/kg/day)	Uncertainty Factor	Non-carcinogenic Effects	Notes for RfD	Slope Factor (mg/kg/day) <sup>-1</sup>	EPA Group	Carcinogenic Effects	Notes for slope factor
<b>Semivolatile organic chemicals</b>								
Acenaphthylene	4.00E-02	-	-	RWQCB	-	-	-	-
Anthracene	3.00E-01	-	-	EPA Region 9	-	-	-	-
Benzo(a)pyrene equivalents	-	-	-	-	3.90E+00	B2	Respiratory tract tumors	Source: Cal EPA
Benzo(g,h,i)perylene	4.00E-02	-	-	RWQCB	-	-	-	-
Dibenzo(a,h)anthracene	-	-	-	-	4.10E+00	B2	Lung carcinoma	Source: Cal EPA
Di-n-butylphthalate	-	-	-	-	-	-	-	-
Fluoranthene	4.00E-02	-	-	EPA Region 9	-	-	-	-
Fluorene	4.00E-02	-	-	EPA Region 9	-	-	-	-
Phenanthrene	4.00E-02	-	-	RWQCB	-	-	-	-
Pyrene	3.00E-02	-	-	EPA Region 9	-	-	-	-
<b>Petroleum hydrocarbons</b>								
Diesel fuel	1.40E-02	-	-	RWQCB	-	-	-	-
Gasoline	1.40E-02	-	-	RWQCB	-	-	-	-
Motor oil	1.40E-02	-	-	RWQCB	-	-	-	-
<b>Metals</b>								
Antimony	-	-	-	-	-	-	-	-
Arsenic	-	-	-	-	1.20E+01	A	Respiratory system tumors	Source: Cal EPA
Barium	1.43E-04	-	-	EPA Region 9	-	-	-	-
Beryllium	5.70E-06	10	Sensitization and progression to berylliosis	IRIS	8.40E+00	B2	Lung tumors	Source: Cal EPA
Cadmium	-	-	-	-	1.50E+01	B1	Lung cancer	Source: Cal EPA
Chromium (total)	-	-	-	-	-	-	-	-
Cobalt	5.70E-06	-	-	EPA Region 9	-	-	-	-
Copper	-	-	-	-	-	-	-	-
Lead	-	-	-	-	4.20E-02	-	-	Source: Cal EPA
Mercury	8.57E-05	30	Neurotoxicity	IRIS	-	-	-	-
Molybdenum	-	-	-	-	-	-	-	-
Nickel	-	-	-	-	9.10E-01	A	Respiratory system tumors	Source: Cal EPA
Selenium	-	-	-	-	-	-	-	-
Silver	-	-	-	-	-	-	-	-
Thallium	-	-	-	-	-	-	-	-
Vanadium	-	-	-	-	-	-	-	-
Zinc	-	-	-	-	-	-	-	-

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**Table 4-2**  
**Oral Reference Doses and Slope Factors**

Chemical	Oral Reference Dose (mg/kg/day)	Uncertainty Factor	Non-carcinogenic Effects	Notes for RfD	Slope Factor (mg/kg/day <sup>-1</sup> )	EPA Group	Carcinogenic Effects	Notes for slope factor
<b>Volatile organic chemicals</b>								
Acetone	9.00E-01	-	-	EPA Region 9	-	-	-	-
Benzene	4.00E-03	300	Decreased lymphocyte count	EPA Region 9	1.00E-01	A	Leukemia	Source: Cal EPA
2-Butanone (Methyl ethyl ketone)	6.00E-01	1,000	Decreased birth weight	IRIS	-	-	-	-
Carbon disulfide	1.00E-01	100	Fetotoxicity	IRIS	-	-	-	-
Chloroform	1.00E-02	1,000	Liver	IRIS	3.10E-02	B2	na	Source: Cal EPA
Chlorormethane	2.60E-02	-	-	EPA Region 9	1.30E-02	D	na	Source: Cal EPA
1,1-Dichloroethane	1.00E-01	-	-	EPA Region 9	5.70E-03	C	na	Source: Cal EPA
1,2-Dichloroethane	3.00E-02	-	-	EPA Region 9	4.70E-02	B2	Lung	Source: Cal EPA
1,1-Dichloroethylene	5.00E-02	100	-	EPA Region 9	-	-	-	-
cis-1,2-Dichloroethylene	1.00E-02	-	-	EPA Region 9	-	-	-	-
trans-1,2-Dichloroethylene	2.00E-02	1,000	Blood	IRIS	-	-	-	-
Ethylbenzene	1.00E-01	1,000	Hepatotoxicity	IRIS	-	-	-	-
Freon 113	3.00E+01	10	Psycho motor impairment	IRIS	-	-	-	-
2-Methylnaphthalene	4.00E-03	1,000	-	IRIS	-	-	-	-
Naphthalene	2.00E-02	3,000	Decreased body weight	IRIS	-	-	-	-
Tetrachloroethylene	1.00E-02	1,000	Blood	IRIS	5.40E-01	na	na	Source: Cal EPA
Toluene	2.00E-01	1000	Altered liver & kidney weights	IRIS	-	-	-	-
1,1,1-Trichloroethane	2.80E-01	-	-	EPA Region 9	-	-	-	-
1,1,2-Trichloroethane	4.00E-03	1,000	Blood	IRIS	7.20E-02	C	na	Source: Cal EPA
Trichloroethylene	3.00E-04	-	-	EPA Region 9	1.30E-02	-	-	Source: Cal EPA
Vinyl Chloride	3.00E-03	30	Liver	IRIS	2.70E-01	A	liver cancer	Source: Cal EPA
m-Xylene	7.00E-01	1,000	Decreased body weight	IRIS	-	-	-	-
o-Xylene	7.00E-01	1,000	Decreased body weight	IRIS	-	-	-	-
Xylenes	7.00E-01	1,000	Decreased body weight	IRIS	-	-	-	-
<b>Semivolatile organic chemicals</b>								
Acenaphthylene	4.00E-02	-	-	RWQCB	-	-	-	-
Anthracene	3.00E-01	3,000	No effects	IRIS	-	-	-	-
Benzo(a)pyrene equivalents	-	-	-	-	1.20E+01	B2	Respiratory tract tumors	Source: Cal EPA
Benzo(g,h,i)perylene	4.00E-02	-	-	RWQCB	-	-	-	-
Dibenzo(a,h)anthracene	-	-	-	-	4.10E+00	B2	Lung carcinoma	Source: Cal EPA
Di-n-butylphthalate	1.00E-01	1,000	Increased mortality	IRIS	-	-	-	-
Fluoranthene	4.00E-02	3,000	Nephropathy	IRIS	-	-	-	-

**Table 4-2**  
**Oral Reference Doses and Slope Factors**

<b>Chemical</b>	<b>Oral Reference Dose (mg/kg/day)</b>	<b>Uncertainty Factor</b>	<b>Non-carcinogenic Effects</b>	<b>Notes for RfD</b>	<b>Slope Factor (mg/kg/day<sup>-1</sup>)</b>	<b>EPA Group</b>	<b>Carcinogenic Effects</b>	<b>Notes for slope factor</b>
Fluorene	4.00E-02	3,000	Decreased red blood cells	IRIS	-	-	-	-
Phenanthrene	4.00E-02	-	-	RWQCB	-	-	-	-
Pyrene	3.00E-02	3,000	Kidney	IRIS	-	-	-	-
<b>Petroleum hydrocarbons</b>								
Diesel Fuel	3.00E-02	-	-	RWQCB	-	-	-	-
Gasoline	3.00E-02	-	-	RWQCB	-	-	-	-
Motor oil	3.00E-02	-	-	RWQCB	-	-	-	-
<b>Metals</b>								
Antimony	4.00E-04	1,000	Increased mortality; altered blood chemistry	IRIS	-	-	-	-
Arsenic	3.00E-04	3	Keratosis & hyperpigmentation	IRIS	9.45E+00	A	-	Source: Cal EPA
Barium	7.00E-02	3	Increased blood pressure	IRIS	-	-	-	-
Beryllium	2.00E-03	300	Small intestinal lesions	IRIS	-	-	-	-
Cadmium	5.00E-04	10	Renal damage	IRIS	3.80E-01	-	-	Source: Cal EPA
Chromium (total)	1.50E+00	100	No effects observed	IRIS	-	-	-	-
Cobalt	2.00E-02	-	-	EPA Region 9	-	-	-	-
Copper	4.00E-02	-	-	EPA Region 9	-	-	-	-
Lead	-	-	-	-	8.50E-03	-	-	Source: Cal EPA
Mercury	3.00E-04	1,000	Immune effects	IRIS	-	-	-	-
Molybdenum	5.00E-03	30	Changes in biological indices	IRIS	-	-	-	-
Nickel	2.00E-02	300	Reduced body weight	IRIS	-	-	-	-
Selenium	5.00E-03	3	Clinical selenosis	IRIS	-	-	-	-
Silver	5.00E-03	3	Argyria	IRIS	-	-	-	-
Thallium	6.60E-05	-	Increased levels of SGOT and LDH	IRIS	-	-	-	-
Vanadium	7.00E-03	-	-	EPA Region 9	-	-	-	-
Zinc	3.00E-01	3	Decreased blood enzyme	IRIS	-	-	-	-

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**Table 5.1**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Inhalation of Volatile Organic Compounds in On-Site Indoor Air**  
**On-Site Commercial/Industrial Worker**

**On-site Indoor Air Sampling Location IA-1**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk (Hazard Quotient)	Theoretical Lifetime Cancer Risks
cis-1,2-Dichloroethylene	ND	ND	NC
Trichloroethylene	1.900	3.72E-02	9.30E-07
<b>Sum of Risks</b>		<b>3.7E-02</b>	<b>9.3E-07</b>

**On-site Indoor Air Sampling Location IA-2**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk (Hazard Quotient)	Theoretical Lifetime Cancer Risks
cis-1,2-Dichloroethylene	1.700	5.84E-03	NC
Trichloroethylene	4.900	9.59E-02	2.40E-06
<b>Sum of Risks</b>		<b>1.0E-01</b>	<b>2.4E-06</b>

**On-site Indoor Air Sampling Location IA-3**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk (Hazard Quotient)	Theoretical Lifetime Cancer Risks
cis-1,2-Dichloroethylene	ND	ND	NC
Trichloroethylene	0.680	1.33E-02	3.33E-07
<b>Sum of Risks</b>		<b>1.3E-02</b>	<b>3.3E-07</b>

**On-site Indoor Air Sampling Location IA-5**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk (Hazard Quotient)	Theoretical Lifetime Cancer Risks
cis-1,2-Dichloroethylene	0.780	2.68E-03	NC
Trichloroethylene	1.800	3.52E-02	8.81E-07
<b>Sum of Risks</b>		<b>3.8E-02</b>	<b>8.8E-07</b>

**On-site Indoor Air Sampling Location IA-6**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk (Hazard Quotient)	Theoretical Lifetime Cancer Risks
cis-1,2-Dichloroethylene	ND	ND	NC
Trichloroethylene	2.400	4.70E-02	1.17E-06
<b>Sum of Risks</b>		<b>4.7E-02</b>	<b>1.2E-06</b>

ND = not detected

NC = not carcinogenic

**Table 5.2**  
**Noncancer and Theoretical Lifetime Cancer Risk Calculations**  
**On-Site Commercial/Industrial Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Noncancer Risks (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Volatile Organic Compounds				
Acetone	0.063	3.42E-08	4.51E-08	1.79E-06
Benzene	0.001	1.20E-07	1.59E-07	1.34E-05
2-Butanone	0.032	2.58E-08	3.40E-08	3.65E-07
Carbon disulfide	0.003	1.39E-08	1.84E-08	3.84E-06
Chloroform	0.003	1.32E-07	1.74E-07	3.25E-05
cis-1,2-Dichloroethylene	0.082	4.01E-06	5.30E-06	9.05E-04
trans-1,2-Dichloroethylene	0.119	2.91E-06	3.84E-06	8.22E-04
Ethylbenzene	0.004	1.84E-08	2.44E-08	7.73E-07
Freon 113	0.015	2.45E-10	3.23E-10	3.56E-07
2-Methylnaphthalene	0.128	1.57E-05	2.07E-05	2.36E-04
Naphthalene	0.162	3.96E-06	5.23E-06	1.40E-03
Tetrachloroethylene	0.009	4.40E-07	5.81E-07	1.13E-04
Toluene	0.011	2.69E-08	3.55E-08	8.06E-06
1,1,1-Trichloroethane	0.009	1.57E-08	2.08E-08	2.07E-06
1,1,2-Trichloroethane	0.001	1.22E-07	1.61E-07	1.24E-05
Trichloroethylene	0.433	7.06E-04	9.32E-04	4.26E-03
Vinyl chloride	0.009	1.42E-06	1.87E-06	9.29E-05
m-Xylene	0.009	6.08E-09	8.03E-09	1.58E-05
o-Xylene	0.016	1.13E-08	1.49E-08	2.92E-05
Semivolatile Organic Compounds				
Acenaphthylene	0.34	4.12E-06	8.16E-06	6.34E-05
Anthracene	0.21	3.47E-07	6.88E-07	3.27E-07
Benzo(a)pyrene equivalents	0.91	NA	NA	NA
Benzo(g,h,i)perylene	0.89	1.08E-05	2.14E-05	3.28E-09
Dibenz(a,h)anthracene	0.27	NA	NA	NA
Di-n-butylphthalate	0.14	6.90E-07	9.11E-07	7.47E-11
Fluoranthene	0.54	6.56E-06	1.30E-05	1.99E-09
Fluorene	0.05	6.35E-07	1.26E-06	8.95E-07
Phenanthrene	0.36	4.37E-06	8.65E-06	5.65E-06
Pyrene	1.17	1.91E-05	3.78E-05	5.79E-09
Petroleum Hydrocarbons				
Diesel fuel	558	9.10E-03	1.20E-02	4.33E-04
Gasoline	2.27	3.70E-05	4.89E-05	3.70E-04
Motor oil	4903	8.00E-02	1.06E-01	3.81E-03

Theoretical Lifetime Cancer Risks		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
1.72E-11	2.27E-11	4.12E-09
NC	NC	NC
NC	NC	NC
1.46E-11	1.93E-11	2.21E-09
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	5.11E-08
8.49E-10	1.12E-09	8.49E-09
NC	NC	NC
NC	NC	NC
1.26E-11	1.66E-11	1.01E-09
9.84E-10	1.30E-09	1.07E-07
4.10E-10	5.42E-10	2.60E-07
NC	NC	NC
NC	NC	NC
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**Table 5.2**  
**Noncancer and Theoretical Lifetime Cancer Risk Calculations**  
**On-Site Commercial/Industrial Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Noncancer Risks (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
<i>Metals</i>				
Antimony	3.40	4.16E-03	5.49E-04	1.26E-06
Arsenic	132	2.15E-01	8.52E-02	NA
Barium	103	7.20E-04	9.50E-05	1.09E-04
Beryllium	0.300	7.34E-05	9.69E-06	7.81E-06
Cadmium	1.20	1.17E-03	1.55E-05	NA
Chromium	45.90	1.50E-05	1.98E-06	NA
Cobalt	15.2	3.72E-04	4.91E-05	3.96E-04
Copper	277	3.39E-03	4.47E-04	NA
Lead	104	NA	NA	NA
Mercury	0.600	9.79E-04	1.29E-04	1.04E-06
Molybdenum	2.60	2.54E-04	3.36E-05	NA
Nickel	37.3	9.12E-04	1.20E-04	NA
Selenium	1.90	1.86E-04	2.45E-05	NA
Silver	0.8	7.88E-05	1.04E-05	NA
Thallium	1.0	7.64E-03	1.01E-03	NA
Vanadium	88	6.12E-03	8.08E-04	NA
Zinc	132.30	2.16E-04	2.85E-05	NA
Risk for Pathway		3.3E-01	2.1E-01	1.3E-02
Sum of Pathway Risks		5.5E-01		

Theoretical Lifetime Cancer Risks		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
2.18E-04	8.63E-05	8.39E-08
NC	NC	NC
NC	NC	1.34E-10
7.97E-08	1.05E-09	9.54E-10
NC	NC	NC
NC	NC	NC
NC	NC	NC
1.55E-07	2.04E-08	2.31E-10
NC	NC	NC
NC	NC	NC
NC	NC	1.80E-09
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
2.2E-04	9.0E-05	5.2E-07
3.1E-04		

NA = Toxicity value not available to assess noncancer risk  
NC = not carcinogenic



**Table 5.3**  
**Noncancer and Theoretical Lifetime Cancer Risk Assessment**  
**On-Site Construction Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Noncancer Risks (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
<i>Volatile Organic Compounds</i>				
Acetone	0.063	1.81E-08	1.62E-08	2.70E-07
Benzene	0.001	6.35E-08	5.69E-08	2.03E-06
2-Butanone	0.032	1.36E-08	1.22E-08	5.51E-08
Carbon disulfide	0.003	7.36E-09	6.60E-09	5.81E-07
Chloroform	0.003	6.98E-08	6.25E-08	4.92E-06
cis-1,2-Dichloroethylene	0.082	2.12E-06	1.90E-06	1.37E-04
trans-1,2-Dichloroethylene	0.119	1.54E-06	1.38E-06	1.24E-04
Ethylbenzene	0.004	9.74E-09	8.73E-09	1.17E-07
Freon 113	0.015	1.29E-10	1.16E-10	5.39E-08
2-Methylnaphthalene	0.128	8.27E-06	7.41E-06	3.56E-05
Naphthalene	0.162	2.09E-06	1.88E-06	2.11E-04
Tetrachloroethylene	0.009	2.33E-07	2.08E-07	1.71E-05
Toluene	0.011	1.42E-08	1.27E-08	1.22E-06
1,1,1-Trichloroethane	0.009	8.30E-09	7.44E-09	3.13E-07
1,1,2-Trichloroethane	0.001	6.46E-08	5.79E-08	1.87E-06
Trichloroethylene	0.433	3.73E-04	3.34E-04	6.44E-04
Vinyl chloride	0.009	7.49E-07	6.72E-07	1.40E-05
m-Xylene	0.009	3.21E-09	2.88E-09	2.39E-06
o-Xylene	0.016	5.94E-09	5.33E-09	4.42E-06
<i>Semivolatile organic compounds</i>				
Acenaphthylene	0.34	2.18E-06	2.93E-06	9.58E-06
Anthracene	0.21	1.83E-07	2.47E-07	4.94E-08
Benzo(a)pyrene equivalents	0.91	NA	NA	NA
Benzo(g,h,i)perylene	0.89	5.72E-06	7.68E-06	2.40E-07
Dibenz(a,h)anthracene	0.27	NA	NA	NA
Di-n-butylphthalate	0.14	3.64E-07	3.27E-07	5.47E-09
Fluoranthene	0.54	3.46E-06	4.65E-06	1.46E-07
Fluorene	0.05	3.35E-07	4.51E-07	1.35E-07
Phenanthrene	0.36	2.31E-06	3.10E-06	8.55E-07
Pyrene	1.17	1.01E-05	1.36E-05	4.24E-07
<i>Petroleum Hydrocarbons</i>				
Diesel fuel	558	4.81E-03	4.31E-03	3.28E-03
Gasoline	2.27	1.96E-05	1.75E-05	1.33E-05
Motor oil	4903	4.22E-02	3.78E-02	3.81E-03

Theoretical Lifetime Cancer Risks		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
2.54E-12	2.28E-12	1.75E-10
NC	NC	NC
NC	NC	NC
2.16E-12	1.94E-12	9.34E-11
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	2.16E-09
1.26E-10	1.13E-10	3.60E-10
NC	NC	NC
NC	NC	NC
1.86E-12	1.67E-12	4.26E-11
1.45E-10	1.30E-10	4.51E-09
6.07E-11	5.44E-11	1.10E-08
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
2.82E-07	3.79E-07	3.86E-09
NC	NC	NC
2.85E-08	3.83E-08	1.20E-09
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC

**Table 5.3**  
**Noncancer and Theoretical Lifetime Cancer Risk Assessment**  
**On-Site Construction Worker Exposed to Chemicals in 0-10' Depth Soil**

Chemical	Exposure Point Concentration (mg/kg)	Noncancer Risks (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
<i>Metals</i>				
Antimony	3.40	2.20E-03	1.97E-04	9.24E-05
Arsenic	132	1.14E-01	3.06E-02	NA
Barium	103	3.80E-04	3.41E-05	7.99E-03
Beryllium	0.300	3.88E-05	3.47E-06	5.72E-04
Cadmium	1.20	6.20E-04	5.56E-06	NA
Chromium	45.90	7.91E-06	7.09E-07	NA
Cobalt	15.2	1.96E-04	1.76E-05	2.90E-02
Copper	277	1.79E-03	1.60E-04	NA
Lead	104	NA	NA	NA
Mercury	0.600	5.17E-04	4.63E-05	7.58E-05
Molybdenum	2.60	1.34E-04	1.20E-05	NA
Nickel	37.3	4.82E-04	4.32E-05	NA
Selenium	1.90	9.82E-05	8.80E-06	NA
Silver	0.8	4.16E-05	3.73E-06	NA
Thallium	1.0	4.03E-03	3.61E-04	NA
Vanadium	88	3.23E-03	2.90E-04	NA
Zinc	132.30	1.14E-04	1.02E-05	NA
Risk for Pathway		1.8E-01	7.4E-02	4.6E-02
Sum of Pathway Risks		3.0E-01		

Theoretical Lifetime Cancer Risks		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
3.22E-05	8.67E-06	1.72E-06
NC	NC	NC
NC	NC	2.74E-09
1.18E-08	1.06E-10	1.96E-08
NC	NC	NC
NC	NC	NC
NC	NC	NC
2.28E-08	2.05E-09	4.75E-09
NC	NC	NC
NC	NC	NC
NC	NC	3.69E-08
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
3.3E-05	9.1E-06	1.8E-06
4.3E-05		

NA = Toxicity value not available to assess noncancer risk  
 NC = not carcinogenic

**Table 5.4**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Off-Site Residents Inhaling Volatile Organic Compounds in Indoor Residential Air**

**3. Hampton Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	1.200	6.67E-02	9.25E-07
Sum of Risks		6.7E-02	9.3E-07
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>2.4E-02</b>	<b>3.3E-07</b>

**4. Hampton Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	0.110	1.07E-03	NC
Trichloroethylene	5.000	2.78E-01	3.86E-06
Sum of Risks		2.8E-01	3.9E-06
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>1.0E-01</b>	<b>1.4E-06</b>

**5. Hampton Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	0.130	1.27E-03	NC
Trichloroethylene	4.300	2.39E-01	3.32E-06
Sum of Risks		2.4E-01	3.3E-06
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>8.7E-02</b>	<b>1.2E-06</b>

**6. Hampton Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	0.430	2.39E-02	3.32E-07
Sum of Risks		2.4E-02	3.3E-07
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>8.6E-03</b>	<b>1.2E-07</b>

**Table 5.4**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Off-Site Residents Inhaling Volatile Organic Compounds in Indoor Residential Air**

**8. Hookston Road**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	0.064	6.25E-04	NC
Trichloroethylene	0.190	1.06E-02	1.47E-07
Sum of Risks		1.1E-02	1.5E-07
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>4.0E-03</b>	<b>5.3E-08</b>

**10. Stimel Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	0.380	2.11E-02	2.93E-07
Sum of Risks		2.1E-02	2.9E-07
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>7.6E-03</b>	<b>1.1E-07</b>

**11. Stimel Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	3.300	1.84E-01	2.54E-06
Sum of Risks		1.8E-01	2.5E-06
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>6.6E-02</b>	<b>9.2E-07</b>

**12. Stimel Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	2.000	1.11E-01	1.54E-06
Sum of Risks		1.1E-01	1.5E-06
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>4.0E-02</b>	<b>5.6E-07</b>

**Table 5.4**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Off-Site Residents Inhaling Volatile Organic Compounds in Indoor Residential Air**

**13. Stimmel Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	0.075	7.32E-04	NC
Trichloroethylene	3.800	2.11E-01	2.93E-06
Sum of Risks		2.1E-01	2.9E-06
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>7.6E-02</b>	<b>1.1E-06</b>

**14. Thames Drive**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	3.100	1.72E-01	2.39E-06
Sum of Risks		1.7E-01	2.4E-06
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>6.2E-02</b>	<b>8.6E-07</b>

**15. Waterloo Court**

Chemical	Air Concentration (ug/m <sup>3</sup> )	Noncancer Risk	Theoretical Lifetime Cancer Risk
1,1-Dichloroethylene	ND	ND	NC
Trichloroethylene	0.420	2.34E-02	3.24E-07
Sum of Risks		2.3E-02	3.2E-07
<b><i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i></b>		<b>8.4E-03</b>	<b>1.2E-07</b>

ND = not detected  
NC = not carcinogenic

**Table 5.5**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(a) Bermuda

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	26.0	8.43E-05	NC
Chloromethane	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	11.0	6.26E-05	NC
cis-1,2-Dichloroethene	24.0	7.78E-04	NC
trans-1,2-Dichloroethene	ND	ND	ND
Tetrachloroethene	ND	ND	ND
Trichloroethene	670.0	1.97E-02	3.54E-07
Vinyl chloride	ND	ND	ND
Sum of Risks		2.1E-02	3.5E-07

***Risk contribution from the on-site Hookston Station Site  
source area to the regional plume***

**7.5E-03**

**1.3E-07**

(b) Bermuda

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	2.2	7.13E-06	NC
Chloromethane	0.8	1.02E-05	3.90E-10
1,1-Dichloroethane	0.2	3.94E-07	7.31E-11
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	1.4	7.96E-06	NC
cis-1,2-Dichloroethene	0.3	8.43E-06	NC
trans-1,2-Dichloroethene	ND	ND	ND
Tetrachloroethene	ND	ND	ND
Trichloroethene	29.0	8.55E-04	1.53E-08
Vinyl chloride	ND	ND	ND
Sum of Risks		8.9E-04	1.6E-08

***Risk contribution from the on-site Hookston Station Site  
source area to the regional plume***

**3.3E-04**

**6.0E-09**

**Table 5.5**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(c) Stimel

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	12.0	3.89E-05	NC
Chloromethane	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	5.0	2.84E-05	NC
cis-1,2-Dichloroethene	17.0	5.51E-04	NC
trans-1,2-Dichloroethene	2.4	3.89E-05	NC
Tetrachloroethene	ND	ND	ND
Trichloroethene	130.0	3.83E-03	6.86E-08
Vinyl chloride	1.0	1.12E-05	2.04E-08
Sum of Risks		4.5E-03	8.9E-08

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***

**1.6E-03**

**3.2E-08**

(d) Stimel

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	ND	ND	ND
Chloromethane	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	4.9	2.79E-05	NC
cis-1,2-Dichloroethene	37.0	1.20E-03	NC
trans-1,2-Dichloroethene	ND	ND	ND
Tetrachloroethene	ND	ND	ND
Trichloroethene	210.0	6.19E-03	1.11E-07
Vinyl chloride	ND	ND	ND
Sum of Risks		7.4E-03	1.1E-07

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***

**2.7E-03**

**4.0E-08**

**Table 5.5**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(e) Stimel

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	ND	ND	ND
Chloromethane	ND	ND	ND
1,1-Dichloroethane	0.3	6.71E-07	1.25E-10
1,2-Dichloroethane	0.3	6.48E-05	1.52E-09
1,1-Dichloroethene	1.7	9.67E-06	NC
cis-1,2-Dichloroethene	12.0	3.89E-04	NC
trans-1,2-Dichloroethene	0.5	7.94E-06	NC
Tetrachloroethene	ND	ND	ND
Trichloroethene	8.0	2.36E-04	4.22E-09
Vinyl chloride	1.5	1.68E-05	3.05E-08
Sum of Risks		7.2E-04	3.6E-08

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***

***3.0E-04***

***1.4E-08***

(f) Gragg Lane

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	3.0	9.72E-06	NC
Chloromethane	0.9	1.12E-05	NC
1,1-Dichloroethane	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	ND	ND	ND
cis-1,2-Dichloroethene	ND	ND	ND
trans-1,2-Dichloroethene	ND	ND	ND
Tetrachloroethene	ND	ND	ND
Trichloroethene	ND	ND	ND
Vinyl chloride	ND	ND	ND
Sum of Risks		2.1E-05	0.0E+00

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***

***2.1E-05***

***0.0E+00***



**Table 5.5**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Inhalation of Volatile Organic Compounds Released from Ground Water During Irrigation**  
**Off-Site Residents**

(g) Thames Drive

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	ND	ND	ND
Chloromethane	ND	ND	ND
1,1-Dichloroethane	1.8	4.17E-06	7.74E-10
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	9.2	5.23E-05	NC
cis-1,2-Dichloroethene	13.0	4.21E-04	NC
trans-1,2-Dichloroethene	ND	ND	ND
Tetrachloroethene	ND	ND	ND
Trichloroethene	500.0	1.47E-02	2.64E-07
Vinyl chloride	ND	ND	ND
Sum of Risks		1.5E-02	2.6E-07

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***

**5.5E-03**

**9.6E-08**

(h) Waterloo

Chemical	Ground water concentration ug/L	Noncancer Risks (Hazard Quotient)	Theoretical Lifetime Cancer Risk
Acetone	12.0	3.89E-05	NC
Chloromethane	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND
1,1-Dichloroethene	4.4	2.50E-05	NC
cis-1,2-Dichloroethene	6.5	2.11E-04	NC
trans-1,2-Dichloroethene	ND	ND	ND
Tetrachloroethene	ND	ND	ND
Trichloroethene	380.0	1.12E-02	2.01E-07
Vinyl chloride	ND	ND	ND
Sum of Risks		1.1E-02	2.0E-07

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***

**4.2E-03**

**7.2E-08**

ND = not detected  
NC = not carcinogenic

**Table 5.6**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

(a) Bermuda

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	26.0	1.26E-06	2.22E-07	3.50E-06
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	11.0	9.57E-06	6.19E-05	2.60E-06
cis-1,2-Dichloroethene	24.0	1.04E-04	4.34E-04	3.23E-05
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	670.0	9.71E-02	7.61E-01	8.21E-04
Vinyl chloride	ND	ND	ND	ND
Risk for Pathway		9.7E-02	7.6E-01	8.6E-04
Sum of Pathway Risks			8.6E-01	

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
7.04E-08	5.51E-07	1.17E-08
ND	ND	ND
7.0E-08	5.5E-07	1.2E-08
	6.3E-07	

**Risk contribution from the on-site Hookston Station Site source area to the regional plume**      **3.1E-01**

**2.3E-07**

(b) Bermuda

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	2.2	1.06E-07	1.88E-08	2.97E-07
Chloromethane	0.8	1.37E-06	1.97E-06	4.25E-07
1,1-Dichloroethane	0.2	7.39E-08	2.70E-07	1.64E-08
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	1.4	1.22E-06	7.88E-06	3.31E-07
cis-1,2-Dichloroethene	0.3	1.13E-06	4.70E-06	3.50E-07
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	29.0	4.20E-03	3.29E-02	3.55E-05
Vinyl chloride	ND	ND	ND	ND
Risk for Pathway		4.2E-03	3.3E-02	3.7E-05
Sum of Pathway Risks			3.7E-02	

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
7.83E-12	2.86E-11	2.43E-12
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
3.05E-09	2.39E-08	5.08E-10
ND	ND	ND
3.1E-09	2.4E-08	5.1E-10
	2.7E-08	

**Risk contribution from the on-site Hookston Station Site source area to the regional plume**      **1.3E-02**

**9.9E-09**

**Table 5.6**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

(c) Stimel

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	12.0	5.80E-07	1.02E-07	1.62E-06
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	5.0	4.35E-06	2.82E-05	1.18E-06
cis-1,2-Dichloroethene	17.0	7.39E-05	3.07E-04	2.29E-05
trans-1,2-Dichloroethene	2.4	5.22E-06	2.17E-05	1.62E-06
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	130.0	1.89E-02	1.48E-01	1.59E-04
Vinyl chloride	1.0	1.45E-05	3.73E-05	4.65E-07
Risk for Pathway		1.9E-02	1.5E-01	1.9E-04
Sum of Pathway Risks			1.7E-01	

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
1.37E-08	1.07E-07	2.28E-09
2.18E-09	5.60E-09	6.76E-10
1.6E-08	1.1E-07	3.0E-09
	1.3E-07	

**Risk contribution from the on-site Hookston Station Site source area to the regional plume**      **6.0E-02**

**4.7E-08**

(d) Stimel

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	4.9	4.26E-06	2.76E-05	1.16E-06
cis-1,2-Dichloroethene	37.0	1.61E-04	6.68E-04	4.99E-05
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	210.0	3.04E-02	2.39E-01	2.57E-04
Vinyl chloride	ND	ND	ND	ND
Risk for Pathway		3.1E-02	2.4E-01	3.1E-04
Sum of Pathway Risks			2.7E-01	

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
2.21E-08	1.73E-07	3.68E-09
ND	ND	ND
2.2E-08	1.7E-07	3.7E-09
	2.0E-07	

**Risk contribution from the on-site Hookston Station Site source area to the regional plume**      **9.7E-02**

**7.1E-08**

**Table 5.6**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

(e) Stimel

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	0.3	1.26E-07	4.61E-07	2.79E-08
1,2-Dichloroethane	0.3	4.06E-07	9.30E-07	2.70E-06
1,1-Dichloroethene	1.7	1.48E-06	9.57E-06	4.02E-07
cis-1,2-Dichloroethene	12.0	5.22E-05	2.17E-04	1.62E-05
trans-1,2-Dichloroethene	0.5	1.07E-06	4.43E-06	3.30E-07
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	8.0	1.16E-03	9.08E-03	9.80E-06
Vinyl chloride	1.5	2.18E-05	5.59E-05	6.97E-07
Risk for Pathway		1.2E-03	9.4E-03	3.0E-05
Sum of Pathway Risks			1.1E-02	

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
1.34E-11	4.88E-11	4.14E-12
1.06E-10	2.44E-10	5.05E-11
NC	NC	NC
NC	NC	NC
NC	NC	NC
NC	NC	NC
8.40E-10	6.58E-09	1.40E-10
3.27E-09	8.41E-09	1.01E-09
4.2E-09	1.5E-08	1.2E-09
	2.1E-08	

**Risk contribution from the on-site Hookston Station Site source area to the regional plume**      **3.8E-03**

**7.8E-09**

(f) Gragg Lane

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	3.0	1.45E-07	2.56E-08	4.04E-07
Chloromethane	0.9	1.51E-06	2.16E-06	4.67E-07
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	ND	ND	ND	ND
cis-1,2-Dichloroethene	ND	ND	ND	ND
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	ND	ND	ND	ND
Vinyl chloride	ND	ND	ND	ND
Risk for Pathway		1.7E-06	2.2E-06	8.7E-07
Sum of Pathway Risks			4.7E-06	

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
ND	ND	ND
0.0E+00	0.0E+00	0.0E+00
	0.0E+00	

**Risk contribution from the on-site Hookston Station Site source area to the regional plume**      **4.7E-06**

**0.0E+00**

(g) Thames Drive

**Table 5.6**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Exposure Volatile Organic Compounds in Ground Water Used to Fill a Swimming Pool**  
**Off-Site Child Resident**

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	ND	ND	ND	ND
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	1.8	7.83E-07	2.86E-06	1.73E-07
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	9.2	8.00E-06	5.18E-05	2.18E-06
cis-1,2-Dichloroethene	13.0	5.65E-05	2.35E-04	1.75E-05
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	500.0	7.25E-02	5.68E-01	6.13E-04
Vinyl chloride	ND	ND	ND	ND

Risk for Pathway      7.3E-02      5.7E-01      6.3E-04  
Sum of Pathway Risks      6.4E-01

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
8.29E-11	3.03E-10	2.57E-11
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
5.25E-08	4.11E-07	8.76E-09
ND	ND	ND

5.3E-08      4.1E-07      8.8E-09  
4.7E-07

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***      **2.3E-01**

**1.7E-07**

(h) Waterloo

Chemical	Ground water concentration ug/L	Noncancer Risk (Hazard Quotients)		
		Ingestion	Dermal contact	Inhalation
Acetone	12.0	5.80E-07	1.02E-07	1.62E-06
Chloromethane	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND
1,1-Dichloroethene	4.4	3.83E-06	2.48E-05	1.04E-06
cis-1,2-Dichloroethene	6.5	2.83E-05	1.17E-04	8.76E-06
trans-1,2-Dichloroethene	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND
Trichloroethene	380.0	5.51E-02	4.32E-01	4.66E-04
Vinyl chloride	ND	ND	ND	ND

Risk for Pathway      5.5E-02      4.3E-01      4.8E-04  
Sum of Pathway Risks      4.9E-01

Theoretical Lifetime Cancer Risk		
Ingestion	Dermal contact	Inhalation
NC	NC	NC
NC	NC	NC
ND	ND	ND
ND	ND	ND
NC	NC	NC
NC	NC	NC
NC	NC	NC
ND	ND	ND
3.99E-08	3.13E-07	6.66E-09
ND	ND	ND

4.0E-08      3.1E-07      6.7E-09  
3.6E-07

***Risk contribution from the on-site Hookston Station Site source area to the regional plume***      **1.8E-01**

**1.3E-07**

ND = not detected  
NC = not carcinogenic

**Table 5.7**  
**Noncancer and Theoretical Lifetime Cancer Risks**  
**Inhalation of Volatile Organic Compounds Volatilizing from Walnut Creek Surface Water**  
**Off-Site Residents**

Chemical	Surface Water Concentration(ug/L)	Noncancer Risk (Hazard Quotients)	Theoretical Lifetime Cancer Risk
cis-1,2-Dichloroethylene	1.4	1.42E-02	NC
Tetrachloroethylene	2.6	1.31E-03	1.09E-06
Trichloroethylene	3.3	2.82E-02	5.06E-07
Sum of Risks		4.4E-02	1.6E-06
<i>Risk contribution from the on-site Hookston Station Site source area to the regional plume</i>		<b>2.9E-02</b>	<b>1.1E-06</b>

NC = not carcinogenic

## **APPENDIX A**

### **ESTIMATE OF SOURCE CONTRIBUTIONS TO HOOKSTON STATION AREA PLUME**

## 1.0 *ESTIMATE OF SOURCE CONTRIBUTIONS TO HOOKSTON STATION AREA PLUME*

The *Remedial Investigation Report* (RIR) (ERM 2004) indicates multiple contributing sources to a regional ground water volatile organic compound (VOC) plume that extends into both the Colony Park neighborhood in Concord and the Fair Oaks community in Pleasant Hill, California. To provide context for the Baseline Human Health Risk Assessment (HRA), an effort was undertaken to estimate the relative contributions from the Hookston Station Site and other sources to this regional ground water plume. In addition, the Regional Water Quality Control Board Order (No. R2-2003-0035, as amended on 15 September 2004) specifically requires conducting the HRA in a manner that “quantitatively evaluate(s) the cumulative risk to human health posed by exposure to contaminants derived from the subject site.” The methodology used to calculate the contribution of the Hookston Station Site-related chemicals to the regional plume is described in this appendix to the HRA.

### 1.1 *CONCEPTUAL METHODOLOGY*

While the Hookston Station Site and the downgradient regional VOC plume have been reasonably well characterized, as documented in the RIR, several off-site contaminant sources in the vicinity of the Hookston Station Site have not been fully investigated. Due to the lack of complete characterization of these other sources, a accurate allocation of contributory mass from each source is not possible. The available regional data set is sufficient, however, to provide a basis for estimation of the Hookston Station Site source contribution to the regional plume.

To understand the contribution of the Hookston Station Site source to the regional plume, ERM developed a three-dimensional computer model of the regional plume. This model assumes a steady-state condition, meaning the plume is not growing or shrinking laterally or vertically over time. This assumption is reasonable for plumes that have developed over many decades in a fairly stable hydrogeologic setting. Chemical molar flux was calculated for cross-sectional transects at various locations in the plume model. “Flux” is a generic term used to describe the mass of chemicals passing across a unit area per unit time. For the purposes of this analysis, “flux” is defined as the mass of chemicals passing across a



defined plane of unit thickness. Time is not a variable, because we have assumed uniform ground water flow velocities throughout the system.

Chemicals migrating within ground water undergo a degradation process (e.g., tetrachloroethene [PCE] degrades to trichloroethene [TCE], then to dichloroethene [DCE]); therefore, mass flux calculations of individual constituents alone are not an accurate means for comparing upgradient (source area) fluxes, where little if any degradation has occurred, with downgradient (residential area) fluxes, where significant degradation may have occurred. The concentrations of chemicals within each transect were therefore converted to their respective formula masses (also known as a “mole”) to determine an overall “molar flux,” representing the number of molecules of chlorinated ethenes within each transect. The areas evaluated for molar flux included:

- Transect A – just downgradient of the Hookston Station Site source area near the 199 Mayhew structure;
- Transect B – along the Hookston Station’s western (upgradient) property boundary, beginning at the site’s northern boundary at Hookston Road, and extending south approximately 650 feet, near the western extent of Transect A; and
- Transect C – across the downgradient portion of the regional plume within the Colony Park neighborhood.

The locations of these transects relative to the shallow (A-Zone) TCE plume and ground water flow directions are shown on Figure A-1. The range of relative contribution of the Hookston Station Site source to the regional plume was estimated by taking the ratio of the molar flux for the Hookston Station transect (Transect A) to that of the downgradient transect (Transect C).

For the purposes of this analysis, we have assumed that the Hookston Station Site is responsible for the molar flux passing across Transect A, and that Transect C represents the molar flux attributable to all contributors to the regional ground water VOC plume.

## 1.2 *DATA INTERPOLATION METHODS*

ERM used a spatial interpolation method (kriging) to estimate the three-dimensional distribution of VOC concentrations in the subsurface beneath the study area. Kriging is a weighted moving average method used to interpolate values from a sample data set onto a regular grid for

contouring. The kriging weights are computed from a variogram, which measures the degree of correlation among sample values in the area as a function of the distance and direction between samples. Given a variogram representative of the area to be estimated, kriging will compute the most accurate estimates possible from the available data, and, as a result, kriging is recognized by the United States Environmental Protection Agency as the best and standard means for interpolation of measured data.

ERM used Mining Visualization System (MVS) software developed by C Tech Development Corporation to interpolate and visualize VOC data from the site. MVS utilizes expert system algorithms to analyze the input data, construct a multidimensional variogram that is a best fit to the data set being analyzed, and then perform kriging within a specified domain.

### **1.3      *DATASET USED FOR KRIGING***

Dissolved-phase ground water concentration data collected between 2001 and 2004 were used to represent the current distribution of VOCs beneath the site. These data are available on Table 8-1 of the RIR. Depth-discrete ground water sample and monitoring well sample data from the A-, B-, and C-zone aquifers were included. For monitoring well samples, only the most recent (July 2004) data set was used. July 2004 data collected by Quest Geosystems Management from the off-site Cuff Property (3343-3355 Vincent Road) were also included.

As a conservative assumption, non-detectable sample results were represented at one-half the laboratory detection limit. Because chemistry data can vary over several orders of magnitude across relatively short distances, the data set was processed to compute the log (base 10) of the parameter value before kriging. Monitoring well data were represented as equally spaced samples distributed within the screened depth interval of the well.

### **1.4      *KRIGING PARAMETERS***

Kriging was performed within a hexahedral finite-element grid, with a spatial extent equal to the convex hull of the data set, or the smallest convex area containing all the data points. The top of the grid was set equal to the water table surface, and the bottom of the grid was set at a constant 105 feet below ground surface (bgs), approximately 2 feet below

the deepest sample point. Adaptive gridding was used to place grid nodes at the exact locations of data points and locally refine the grid in the areas or volumes surrounding the data points. Adaptive gridding ensures that the data minimum and maximum in the gridded model match the sample data, and provides greater fidelity in defining data trends in regions with high gradients.

Kriging was performed using a default horizontal-to-vertical anisotropy ratio of 10, which allows data points in a horizontal direction to influence the kriged value at a nearby node 10 times more than data points an equal distance away in the vertical direction. This ratio is considered appropriate given the hydrogeologic characteristics of the site, which is underlain by unconsolidated alluvial deposits. The kriged three-dimensional concentration distribution was then calculated for each of six VOCs of concern:

- PCE
- TCE
- cis-1,2-DCE
- trans-1,2-DCE
- 1,1-DCE
- Vinyl chloride

## **1.5      *GENERATION OF TRANSECTS FOR MOLAR FLUX CALCULATIONS***

To calculate average VOC concentrations and estimate molar VOC fluxes within specified portions of the kriged data field, three cross-sectional slice planes were positioned within the kriged model along Transects A, B, and C, as shown on Figure A-1 and described above. The transects cut the data field from the top of the grid (water table surface) to the bottom (approximately 105 feet bgs). Each transect represents a thin, quadrilateral slice of the kriged data field approximately 1 foot thick.

The average VOC concentration within each transect was calculated by MVS, and the results are summarized on Table A-1. This table also includes estimates of the mass and number of moles of VOCs contained within each 1-foot slice. For the purposes of this analysis, we have assumed uniform ground water flow velocities perpendicular to the slice planes. The total moles presented in Table A-1 are therefore equivalent to the VOC molar flux across each surface.

A contribution percentage has been estimated by comparing the molar flux of chlorinated ethenes across these three transects. As shown in Table A-1, if we compare the Hookston source area (Transect A) to the total flux of chemicals migrating across Transect C, the Hookston Station Site chemicals represent approximately 36% of the total downgradient flux. This Hookston Station flux allocation has been incorporated into the risk calculations within the HRA. For comparison, the Vincent Road PCE source contribution (Transect A molar flux divided by Transect C molar flux) is approximately 18% of the total downgradient flux.

It is important to note that the above calculations are preliminary and are based on the sources of VOCs that are known to exist. Due to the lack of complete characterization of other known or suspected sources, and the potential that additional, but currently unknown, sources exist within this area, a precise allocation of mass from each source is not possible. Although we have assumed steady state conditions, there are loss terms that are not accounted for in this analysis, including volatilization of chemicals from the water table and dilution due to rainwater infiltration. The mass misbalance between Transects A and B with Transect C ( $C > A + B$ ) could represent an additional source term between these transect lines. The most likely location for an additional source term is in the vicinity of MW-14A (Figure A-1), where some of the highest concentrations are observed within the plume. This location is off the Hookston Station Site, and is just north of former machine shops and current commercial businesses along Bancroft Road. None of the parcels south of MW-14A have been investigated to date. Access to one parcel was sought during the Remedial Investigation fieldwork, but the property owner denied access.

Significant concentrations of VOCs have also been identified in ground water along Estand Way (just north of Vincent Road) and within the Fair Oaks neighborhood. Unidentified sources to ground water contamination in this area (which are clearly not associated with historic releases at the Hookston Station Site) could also be affecting the overall chemical flux within the downgradient residential area. Additional investigation of Bancroft Road area and the Estand Way/Fair Oaks area by the appropriate responsible parties will be necessary to quantify these source contributions to the regional VOC plume.

**Table A-1 Molar Flux Calculations**  
**Combined A,B,C Zone Ground Water**  
**Hookston Station, Pleasant Hill, CA**

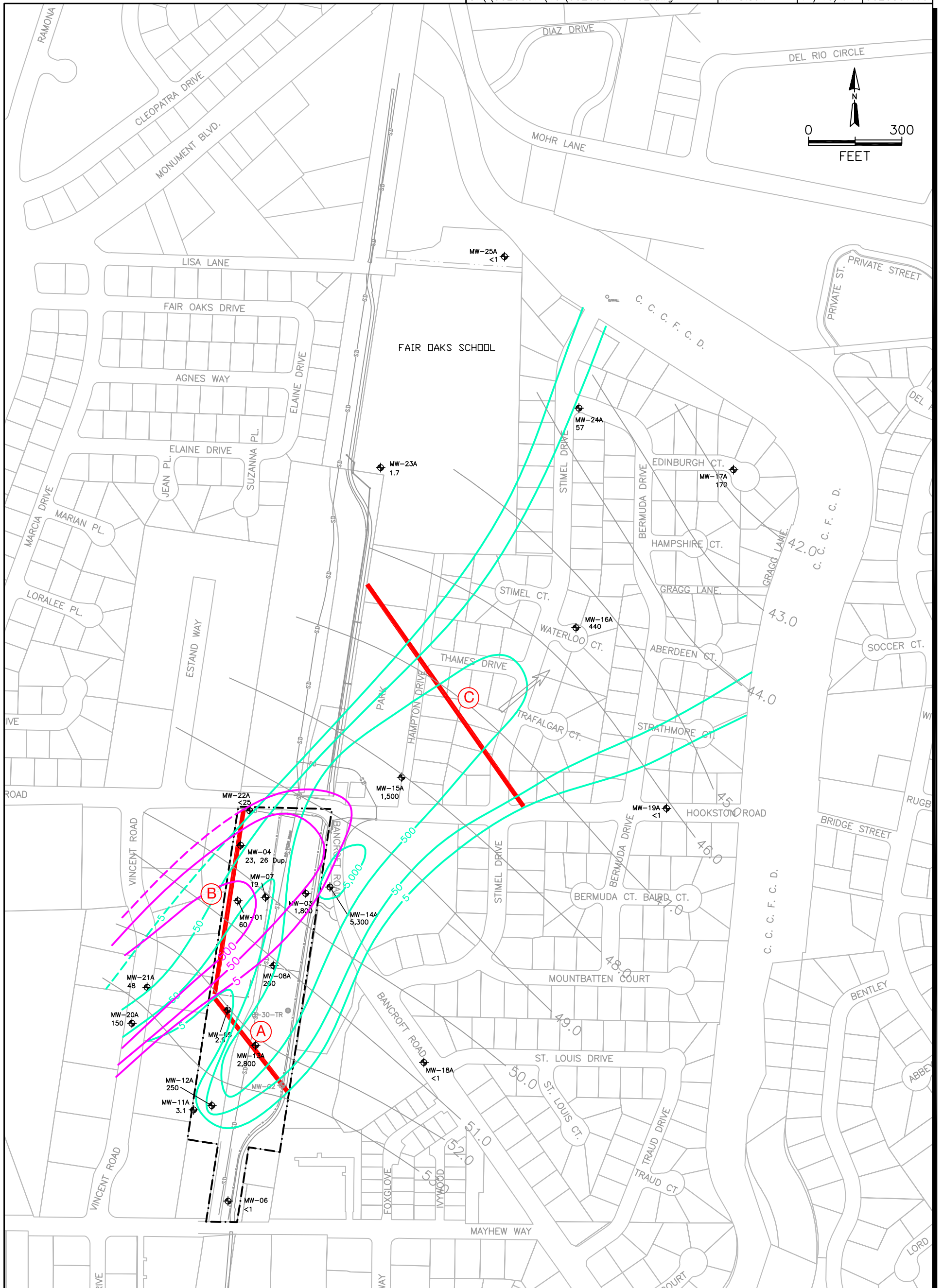
	Surface #1 (Transect A)	Surface #2 (Transect B)	Surface #3 (Transect C)
Width (ft):	356	627	902
Depth (ft):	89	89	89
Surface Area (sq ft):	31,684	55,803	80,278
Unit Volume of Surface (L)*:	897,191	1,580,165	2,273,220
Average PCE (ug/L):	3.0	10.8	1.4
PCE mass on surface (g):	2.7	17.1	3.2
Moles of PCE on Surface (mol):	0.016	0.103	0.019
Average TCE (ug/L):	110.9	25.1	141.6
TCE mass on surface (g):	99.5	39.7	321.9
Moles of TCE on Surface (mol):	0.757	0.302	2.450
Average cis-1,2-DCE (ug/L):	11.5	1.3	4.8
cis-1,2-DCE mass on surface (g):	10.3	2.1	10.9
Moles of cis-1,2-DCE on Surface (mol):	0.106	0.021	0.113
Average trans-1,2-DCE (ug/L):	2.4	0.9	1.7
trans-1,2-DCE mass on surface (g):	2.2	1.4	3.9
Moles of trans-1,2-DCE on Surface (mol):	0.022	0.015	0.040
Average 1,1-DCE (ug/L):	8.4	3.1	8.1
1,1-DCE mass on surface (g):	7.5	4.9	18.4
Moles of 1,1-DCE on Surface (mol):	0.078	0.051	0.190
Average Vinyl Chloride (ug/L):	2.7	0.9	1.4
VC mass on surface (g):	2.4	1.4	3.2
Moles of VC on Surface (mol):	0.039	0.023	0.051
Total Moles of PCE, TCE, c-1,2-DCE, 1,1-DCE, and VC*	1.019	0.514	2.862
Hookston Upgradient Onsite Source Contribution (Transect A to Transect C)**	36%		
Vincent Road PCE Source Contribution (Transect B to Transect C)***		18%	

Nominal VOC concentrations based on mathematical interpolation (at 60% confidence) of RI data from 2001-2004.

\* = Surface Area x 1 foot thickness

\*\* = This is the ratio of the flux associated with the upgradient onsite Hookston source and downgradient regional plume .

\*\*\* = This is the ratio of the flux associated the upgradient (Vincent Road vicinity) source(s) and the flux within the downgradient regional plume.



### LEGEND

Monitoring Well location  
TCE concentration ( $\mu\text{g/L}$ )

TCE in ground water contour, dashed where inferred;  
Data collected second quarter 2004

PCE in ground water contour, dashed where inferred;  
Data collected second quarter 2004

Ground water elevation contour, A-Zone, July 2004  
(feet above mean sea level)

Site Boundary

VOC Molar Flux Transect

Figure A-1  
*VOC Molar Flux Transect Locations*  
*Hookston Station Project*  
*Pleasant Hill, California*

## **APPENDIX B**

### **CALCULATION OF AIR CONCENTRATIONS FOR CHEMICALS RELEASED FROM SOIL**

This appendix describes the calculation of volatile chemical concentrations in ambient air resulting from volatilization from on-site soils. The calculated air concentrations were used to estimate inhalation exposures for commercial/industrial workers and construction workers. The discussion is organized as follows:

- Calculation of on-site air concentrations for chemicals volatilized from soil.
- Calculation of the volatilization factors (VFs) and chemical flux estimates used in estimating air concentrations in the preceding calculations.

#### Calculation of Air Concentrations

On-site air concentrations of the chemicals of concern were calculated by dividing the soil concentration of the chemical (in mg/kg) by the appropriate volatilization factor (VF). The resultant concentrations were then used to assess exposure and risk for on-site receptors (i.e., on-site worker, trespasser, and the construction worker).

$$\text{On-site air concentration (mg/m}^3\text{) for volatile chemicals} = \frac{\text{Soil concentration (mg / kg)}}{\text{VF (m}^3 \text{ / kg)}}$$

#### Calculation of Volatilization Factors (VFs)

VOCs having a Henry's Law Constant greater than  $10^{-5}$  (atm-m<sup>3</sup>/mol) and a molecular weight less than 200. In addition, TPH as gasoline was also considered to be a VOC.

Several of the terms used in the calculation of the VF are chemical-specific; they were derived from physical and chemical information obtained from the SFRWQCB guidance (SFRWQCB, 2003) and USEPA's Soil Screening Guidance: Technical Background Document (USEPA, 1996). In those cases where air diffusivities ( $D_i$ ) and Henry's Law Constants were not provided in the SFRWQCB or USEPA reference,  $D_i$ 's and Henry's Law Constants were derived based on analogy to structurally similar chemicals.

The equation presented below was used to calculate VFs for the volatile chemicals of concern:

$$\text{VF (m}^3\text{/kg)} = (\text{Q/C}) \times [(3.14 \times D_A \times T)^{0.5} / (2 \times d_b \times D_A)] \times 10^{-4} \text{ (m}^2\text{/cm}^2\text{)}$$

where

$$D_A = [(\theta_a^{10/3} D_i H' + \theta_w^{10/3} D_w) / n^2] / (d_b K_d + \theta_w + \theta_a H')$$

and

$D_A$  = Apparent diffusivity, cm<sup>2</sup>/s

$Q/C$  = Inverse of the mean concentration of the Hookston Station site (g/m<sup>2</sup>-sec per kg/m<sup>3</sup>) [A 30-acre source area with a  $Q/C$  value of 46.06 for the San Francisco area; USEPA, 1996]



- T = Exposure interval, seconds [equal to the exposure duration expressed in second; commercial industrial worker, 7.88E+08 sec; construction worker, 2.21E+08 sec]
- $\rho_b$  = Dry soil bulk density, g/cm<sup>3</sup> [1.5 g/cm<sup>3</sup>, USEPA, 1996]
- $\phi_a$  = Air filled porosity,  $L_{air}/L_{soil}$  [0.19; site-specific]
- n = Total soil porosity,  $L_{pore}/L_{soil}$  [0.28; site-specific]
- $\phi_w$  = Water filled soil porosity,  $L_{water}/L_{soil}$  [0.0.09; site-specific]
- $\rho_s$  = Soil particle density, g/cm<sup>3</sup> [2.65 g/cm<sup>3</sup>; USEPA, 1996]
- $D_i$  = Diffusivity in air, cm<sup>2</sup>/sec [chemical-specific]
- H = Henry's Law Constant, atm-m<sup>3</sup>/mol [chemical-specific]
- H' = Henry's Law Constant, dimensionless [chemical-specific]
- $D_w$  = Diffusivity in water, cm<sup>2</sup>/sec [chemical-specific]
- $K_d$  = Soil water partition coefficient, cm<sup>3</sup>/g [chemical-specific;  $K_d = K_{oc} \times f_{oc}$ ]
- $K_{oc}$  = Soil organic carbon-water partition coefficient, cm<sup>3</sup>/g [chemical-specific]
- $f_{oc}$  = Fraction organic carbon in soil, g/g [0.006; USEPA, 1996]

Chemical-specific inputs and calculated VFs and air concentrations are presented in the attached tables.

### Model Inputs for Volatile Chemicals

Volatile Chemicals	Koc (cm <sup>3</sup> /g)	Diffusivity in air (cm <sup>2</sup> /sec)	Diffusivity in water (cm <sup>2</sup> /sec)	Henrys Law (atm-m <sup>3</sup> /mol)	Calculated Apparent Diffusivity (cm <sup>2</sup> /sec)
Acetone	5.75E-01	0.124	1.14E-05	1.59E-03	9.95E-05
Benzene	5.90E+01	0.088	9.80E-06	2.28E-01	2.10E-03
2-Butanone	4.50E+00	0.0895	9.80E-06	1.12E-03	4.15E-05
Carbon disulfide	4.57E+01	0.104	1.00E-05	1.24E+00	1.11E-02
Chloroform	3.98E+01	0.104	1.00E-05	1.50E-01	2.21E-03
cis-1,2-DCE	3.55E+01	0.0736	1.13E-05	1.67E-01	1.86E-03
trans-1,2-DCE	5.25E+01	0.0707	1.19E-05	3.85E-01	2.91E-03
Ethylbenzene	3.63E+02	0.075	7.80E-06	3.23E-01	5.39E-04
Freon 113	1.60E+02	0.029	8.10E-06	2.10E+01	6.36E-03
2-Methylnaphthalene	7.20E+02	0.059	7.50E-06	1.19E-02	8.27E-06
Naphthalene	1.19E+03	0.059	7.50E-06	1.98E-02	8.39E-06
Tetrachloroethylene	1.55E+02	0.072	8.20E-06	7.54E-01	2.41E-03
Toluene	1.82E+02	0.087	8.60E-06	2.72E-01	9.90E-04
1,1,1-Trichloroethane	1.10E+02	0.078	8.80E-06	7.05E-01	3.21E-03
1,1,2-Trichloroethane	5.01E+01	0.078	8.80E-06	3.74E-02	3.72E-04
Trichloroethylene	1.66E+02	0.079	9.10E-06	4.22E-01	1.48E-03
Vinyl chloride	1.86E+01	0.106	1.23E-06	1.11E+00	1.46E-02
m-Xylene	4.07E+02	0.07	7.80E-06	3.01E-01	4.22E-04
o-Xylene	4.07E+02	0.07	7.80E-06	3.01E-01	4.22E-04
Acenaphthylene	2.50E+03	0.0421	7.69E-06	1.45E-03	2.14E-07
Anthracene	2.35E+04	0.0324	7.74E-06	6.50E-05	1.13E-09
Fluorene	1.38E+04	0.0363	7.88E-06	7.70E-05	2.37E-09
Phenanthrene	1.40E+04	0.0608	7.88E-06	3.93E-05	2.09E-09
Gasoline	5.00E+03	0.088	7.50E-06	7.20E-04	1.11E-07

### Calculated VFs and Air Concentrations for Volatile Chemicals

Volatile Chemicals	Soil conc (mg/kg)	Commercial/ Industrial Worker VF (m <sup>3</sup> /kg)	VOC air conc C/I worker (mg/m <sup>3</sup> )	Construction Worker VF (m <sup>3</sup> /kg)	VOC air conc Const worker (mg/m <sup>3</sup> )
Acetone	0.0629	7.66E+03	8.21E-06	4.05E+03	1.55E-05
Benzene	0.001	1.67E+03	5.90E-07	8.82E+02	1.11E-06
2-Butanone	0.0316	1.19E+04	2.66E-06	6.28E+03	5.03E-06
Carbon disulfide	0.0029	7.26E+02	3.93E-06	3.84E+02	7.42E-06
Chloroform	0.0027	1.62E+03	1.66E-06	8.60E+02	3.14E-06
cis-1,2-DCE	0.082	1.77E+03	4.63E-05	9.38E+02	8.74E-05
trans-1,2-DCE	0.119	1.42E+03	8.40E-05	7.50E+02	1.59E-04
Ethylbenzene	0.0038	3.29E+03	1.15E-06	1.74E+03	2.16E-06
Freon 113	0.015	9.58E+02	1.57E-05	5.07E+02	2.96E-05
2-Methylnaphthalene	0.128	2.66E+04	4.82E-06	1.41E+04	9.10E-06
Naphthalene	0.162	2.64E+04	6.14E-06	1.40E+04	1.16E-05
Tetrachloroethylene	0.009	1.56E+03	5.79E-06	8.23E+02	1.09E-05
Toluene	0.011	2.43E+03	4.53E-06	1.29E+03	8.56E-06
1,1,1-Trichloroethane	0.009	1.35E+03	6.67E-06	7.14E+02	1.26E-05
1,1,2-Trichloroethane	0.001	3.96E+03	2.53E-07	2.10E+03	4.77E-07
Trichloroethylene	0.433	1.99E+03	2.18E-04	1.05E+03	4.12E-04
Vinyl chloride	0.0087	6.32E+02	1.38E-05	3.34E+02	2.60E-05
m-Xylene	0.0087	3.72E+03	2.34E-06	1.97E+03	4.42E-06
o-Xylene	0.0161	3.72E+03	4.33E-06	1.97E+03	8.18E-06
Acenaphthylene	0.337	1.65E+05	2.04E-06	8.75E+04	3.85E-06
Anthracene	0.213	2.27E+06	9.39E-08	1.20E+06	1.77E-07
Fluorene	0.0519	1.57E+06	3.31E-08	8.30E+05	6.25E-08
Phenanthrene	0.357	1.67E+06	2.14E-07	8.85E+05	4.03E-07
Gasoline	2.27	2.29E+05	9.90E-06	1.21E+05	1.87E-05

## **APPENDIX C**

### **CALCULATION OF EXPOSURES TO CHEMICALS OF POTENTIAL CONCERN IN GROUND WATER USED FOR IRRIGATION AND FILLING SWIMMING POOLS**

## **Irrigation Exposure Scenario**

Shallow ground water is assumed to be used to irrigate a yard. In the irrigation scenario, residents are assumed to water a residential lawn during the warmest weeks of the year (18 weeks). Volatile organic compounds are assumed to completely volatilize over an 8 hour period starting with the onset of irrigation. Residents are assumed to be exposed over the entire 8 hour volatilization period by inhaling the volatilizing VOCs. Such a scenario is likely to occur over nighttime hours when residents are at home and evaporation of the irrigation water is efficiently minimized.

Chemicals of potential concern (COPCs) for the irrigation scenario were selected as those chemicals that were detected in ground water from 8 private wells located in the Hookston Station area.

The following assumptions were used to estimate VOC emissions from ground water used for irrigation.

### **Assumptions**

#### **Amount of ground water for irrigation**

Conservatively, 7.62 cm (3 inches) of water per week are assumed to be needed for lawn irrigation weekly. According to Maddaus and Mayer ("Splash or Sprinkle? Comparing the Water Use of Swimming Pools and Irrigated Landscapes", undated), annual irrigation water use in arid climates (Boulder, Denver, San Diego, Phoenix, Tempe, Scottsdale, Walnut Valley, Las Virgenes, and Lompoc) ranged from 20.8 to 45.4 inches per year. Given the assumptions below (18 weeks of irrigation at 3 inches per week), annual irrigation with ground water is assumed to be 54 inches per year. This is a reasonably conservative estimate of the amount of ground water used to irrigate lawns in the Hookston Station area.

#### *Number of weeks of lawn irrigation*

Lawn irrigation is assumed to occur over 18 weeks (May 15 through September 15).

#### *Number of irrigation events during the irrigation season*

Lawns are assumed to be irrigated every other day for 18 weeks for 63 irrigation events per season or 3.5 events per week.

#### **Area irrigated**

The USEPA default residential exposure unit of 0.5 acre (20,235,000 cm<sup>2</sup>) is assumed.

*Total amount of water used per irrigation event*

$$= (7.62 \text{ cm per week} / 3.5 \text{ irrigation events per week}) \times 20,235,000 \text{ cm}^2 \times 0.001 \text{ cm}^3/\text{L} = 44,100 \text{ L}$$

### **Rate of volatile emissions from ground water**

VOCs are assumed to entirely volatilize within 8 hours.

### **Emission Calculations**

The rate of volatilization of the VOCs from ground water used for irrigation is calculated according to the formula below:

$$\text{VOC concentration in water (ug/L)} \times 1\text{E-}6 \text{ g/ug} \times 44,100 \text{ L/8 hours} \times (8 \text{ hours}/28,800 \text{ seconds}) \times (1/20,250,000 \text{ cm}^2) = \text{Average rate of VOC flux (g/cm}^2\text{/sec)}$$

#### *Calculation of Air Concentrations*

The residential VOC air concentrations of resulting from emission from using ground water/surface water for irrigation is calculated according to the formula:

$$C_{air} = \frac{\text{Rate of VOC flux} \times 10^4 \text{ cm}^2 / \text{m}^2}{Q / C \times 10^{-6} \text{ kg} / \text{mg}}$$

where:

CA= Concentration in air; ug/m<sup>3</sup>

Rate of VOC flux = calculated value, g/cm<sup>2</sup>/sec

If it is assumed that the VOC concentration in ground water is 1 ug/L, the calculated average rate of flux of VOCs during one irrigation event is calculated as

$$1 \text{ ug/L} \times 1\text{E-}6 \text{ g/ug} \times 44,100 \text{ L/8 hours} \times (8 \text{ hours}/28,800 \text{ seconds}) \times (1/20,250,000 \text{ cm}^2) = 7.56 \times 10^{-14} \text{ g/cm}^2\text{-s}$$

Q/C = inverse concentration factor for air dispersion for a 0.5 acre property in San Francisco (89.53 g/m<sup>2</sup>-s per kg/m<sup>3</sup>; USEPA, 1996)

Using the above equation and the assumptions discussed, the average air concentration after an irrigation event (assumed to be 8 hours) is 0.00846 ug/m<sup>3</sup>. From this information, an irrigation specific volatilization factor can be calculated. This volatilization factor (VF<sub>irr</sub>) is 0.00844 ug/m<sup>3</sup> per 1 ug/L or 0.00846 L/m<sup>3</sup>. This value is used in calculating inhalation exposures to the chemicals of potential concern in ground water used for irrigation.

Calculated air concentrations by Hookston Station area location are presented in the table below.

**Modeled Air Concentrations of VOCs from Irrigation Activity**

Volatile Chemical	(a) Bermuda		(b) Bermuda		(c) Stimel		(d) Stimel	
	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc
Acetone	26.0	0.2200	2.2	0.0186	12.0	0.1010	ND	NC
Chloromethane	ND	NC	0.8	0.0069	ND	NC	ND	NC
1,1-Dichloroethane	ND	NC	0.2	0.0014	ND	NC	ND	NC
1,2-Dichloroethane	ND	NC	ND	NC	ND	NC	ND	NC
1,1-Dichloroethene	11.0	0.0930	1.4	0.0118	5.0	0.0423	4.9	0.0414
cis-1,2-Dichloroethene	24.0	0.2030	0.3	0.0022	17.0	0.1440	37.0	0.3130
trans-1,2-Dichloroethene	ND	NC	ND	NC	2.4	0.0203	ND	NC
Tetrachloroethene	ND	NC	ND	NC	ND	NC	ND	NC
Trichloroethene	670.0	5.6600	29.0	0.2450	130.0	1.1000	210.0	1.7700
Vinyl chloride	ND	NC	ND	NC	1.0	0.0085	ND	NC

Volatile Chemical	(e) Stimel		(f) Gragg Lane		(g) Thames Dr		(h) Waterloo	
	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc
Acetone	ND	NC	3.0	0.0254	ND	NC	12.0	0.1010
Chloromethane	ND	NC	0.9	0.0076	ND	NC	ND	NC
1,1-Dichloroethane	0.3	0.0025	ND	NC	1.8	0.0152	ND	NC
1,2-Dichloroethane	0.3	0.0024	ND	NC	ND	NC	ND	NC
1,1-Dichloroethene	1.7	0.0144	ND	NC	9.2	0.0777	4.4	0.0372
cis-1,2-Dichloroethene	12.0	0.1010	ND	NC	13.0	0.1100	6.5	0.0549
trans-1,2-Dichloroethene	0.5	0.0041	ND	NC	ND	NC	ND	NC
Tetrachloroethene	ND	NC	ND	NC	ND	NC	ND	NC
Trichloroethene	8.0	0.0676	ND	NC	500.0	4.2300	380.0	3.2100
Vinyl chloride	1.5	0.0127	ND	NC	ND	NC	ND	NC

GW conc = ground water concentration in ug/L

Modeled air conc = modeled air concentration from irrigation activities in ug/m<sup>3</sup>

ND = not detected

NC = not calculated, chemical not detected in ground water

## Swimming Exposure Scenario

In the swimming exposure scenario, a resident is assumed to fill a backyard pool with ground water containing the chemicals of potential concern. Chemicals of potential concern (COPCs) for the swimming exposure scenario were selected as those chemicals that were detected in ground water samples from 8 private wells in the Hookston Station area.

Exposure to the chemicals of potential concern in swimming pool water was assumed to occur via skin uptake during swimming, inhalation of volatilizing COPCs, and ingestion of pool water.

Pool filling was assumed to occur once per season. Ground water was also assumed to be used to make up for losses resulting from evaporation and splashing.

The swimming season is assumed to last 18 weeks (approximately May 15 through September 15) or 126 days. During this time, a child is assumed to swim 6 days per week for 1 hour per day.

### *Concentration of the COPCs in Swimming Pool Water*

Due to their volatile nature, losses of the COPCs via volatilization are accounted for by assuming an average rate of volatilization in which 50% of the chemical in the pool water will volatilize with 3.5 days. A typical backyard swimming pool is 30 feet long x 15 feet wide x 5 feet deep and would contain approximately 2250 cubic feet or 64,000 liters of water. Based on estimates for the Sacramento area prepared by the California Spa and Pool Industry Energy, Codes and Legislative Council (SPEC, 2002), a pool this size would require approximately 1000 L per day of water to replenish the pool (from water losses caused by evaporation, splashing, etc.).

Assuming that 1000 L per day of ground water are needed to replenish the pool, the seasonal average COPC concentration the pool over 126 days is calculated below.

Assume 3.5 day half life (volatilization rate constant of  $0.198 \text{ days}^{-1}$ )

Assume ground water concentration is 1 ug/L

Assume pool contains 64,000 L of ground water

The first day after filling, the concentration of COPC in pool after 24 hours of original filling =  $1 \text{ mg/L} \times e^{(-0.198 \times 1)} = 0.82 \text{ mg/L}$  at a volume of 63,000L

Add to this 1000 L containing 1 ug/L- what is the adjusted COPC concentration in pool water?

(Concentration in pool x 63,000 L) + (1 ug/L x 1000 L) divided by 64,000 L

$$= 0.823 \text{ ug/L} \times e^{(-0.198 \times 1)} = 0.675 \text{ ug/L at a volume of 63,000L}$$

Add to this 1000 L containing 1 ug/L and the adjusted Day 2 COPC concentration in pool water is calculated as (0.675 ug/L x 63,000 L) + (1 ug/L x 1000 L) divided by 64,000 L = 0.68 ug/L. This calculation was repeated for 30 days. It was determined that the concentration declines to 0.083 mg/L after about 30 days and remains fairly constant from Day 30 through Day 126. The average COPC concentration in water over the 126 day swimming season is 0.122 ug/L. Based on these calculations, a swimming pool loss factor of 0.122 (0.122 ug/L divided by 1 ug/L) was calculated.

The equation used to calculate the dermally absorbed dose of the chemicals of concern in swimming pool water requires the calculation of a chemical-specific dermally absorbed dose through the skin. This value is called the  $DA_{event}$ .

For tetrachloroethylene and trichloroethylene, the  $DA_{event}$  was calculated using the following formula:

$$2 \times K_p \times CW \times 0.001 \text{ mg / ug} \times \text{swimming pool loss factor} \times \frac{0.001 \text{ mg}}{\text{microgram}} \times \frac{L}{1000 \text{ cm}^3} \times \sqrt{\frac{6 \times \tau \times t_{event}}{\pi}}$$

For all other VOCs of potential concern,  $DA_{event}$  was calculated using the formula presented below:

$$K_p \times CW \times 0.001 \text{ mg / ug} \times \text{swimming pool loss factor} \times \left[ \frac{t_{event}}{1 + B} \right] + 2\tau \times \left[ \frac{1 + 3B}{1 + B} \right]$$

where:

$DA_{event}$  = dermal dose absorbed through the skin per exposure event (mg/cm<sup>2</sup>)

$K_p$  = dermal permeability coefficient from Exhibit B-3 of USEPA, 2001 (cm/hr)

CW = concentration in water (ug /L)

$\tau$  = Chemical-specific; from Exhibit B-3 of USEPA, 2001 (hours)

$t_{event}$  = hours of exposure to water per event (1 hour)

$\pi$  = 3.14

The values of  $K_p$ ,  $\tau$ , and the calculated  $DA_{event}$  by Hookston Station location are presented in the tables below.



### Values for $K_p$ and $\tau$ for the Chemicals of Potential Concern

Chemical	$K_p$	$\tau$
Acetone	0.0006	0.27
Chloromethane	0.0033	0.2
1,1-Dichloroethane	0.0067	0.38
1,2-Dichloroethane	0.0042	0.38
1,1-Dichloroethene	0.012	0.37
cis-1,2-Dichloroethene	0.0077	0.37
trans-1,2-Dichloroethene	0.0077	0.37
Tetrachloroethene	0.033	0.91
Trichloroethene	0.012	0.58
Vinyl chloride	0.0056	0.24

### Modeled $DA_{event}$ by Hookston Station Site Location

Volatile Chemical	(a) Bermuda		(b) Bermuda		(c) Stimel		(d) Stimel	
	GW conc	$DA_{event}$	GW conc	$DA_{event}$	GW conc	$DA_{event}$	GW conc	$DA_{event}$
Acetone	26.0	2.78E-09	2.2	2.35E-10	12.0	1.28E-09	ND	NC
Chloromethane	ND	NC	0.8	4.62E-10	ND	NC	ND	NC
1,1-Dichloroethane	ND	NC	0.2	2.45E-10	ND	NC	ND	NC
1,2-Dichloroethane	ND	NC	ND	NC	ND	NC	ND	NC
1,1-Dichloroethene	11.0	2.80E-08	1.4	3.57E-09	5.0	1.27E-08	4.9	1.25E-08
cis-1,2-Dichloroethene	24.0	3.92E-08	0.3	4.25E-10	17.0	2.78E-08	37.0	6.05E-08
trans-1,2-Dichloroethene	ND	NC	ND	NC	2.4	3.92E-09	ND	NC
Tetrachloroethene	ND	NC	ND	NC	ND	NC	ND	NC
Trichloroethene	670.0	2.07E-06	29.0	8.94E-08	130.0	4.01E-07	210.0	6.47E-07
Vinyl chloride	ND	NC	ND	NC	1.0	1.01E-09	ND	NC

Volatile Chemical	(e) Stimel		(f) Gragg Lane		(g) Thames Dr		(h) Waterloo	
	GW conc	$DA_{event}$	GW conc	$DA_{event}$	GW conc	$DA_{event}$	GW conc	$DA_{event}$
Acetone	ND	NC	3.0	3.21E-10	ND	NC	12.0	1.28E-09
Chloromethane	ND	NC	0.9	5.07E-10	ND	NC	ND	NC
1,1-Dichloroethane	0.3	4.17E-10	ND	NC	1.8	2.59E-09	ND	NC
1,2-Dichloroethane	0.3	2.53E-10	ND	NC	ND	NC	ND	NC
1,1-Dichloroethene	1.7	4.33E-09	ND	NC	9.2	2.34E-08	4.4	1.12E-08
cis-1,2-Dichloroethene	12.0	1.96E-08	ND	NC	13.0	2.13E-08	6.5	1.06E-08
trans-1,2-Dichloroethene	0.5	8.01E-10	ND	NC	ND	NC	ND	NC
Tetrachloroethene	ND	NC	ND	NC	ND	NC	ND	NC
Trichloroethene	8.0	2.47E-08	ND	NC	500.0	1.54E-06	380.0	1.17E-06
Vinyl chloride	1.5	1.52E-09	ND	NC	ND	NC	ND	NC

GW conc = ground water concentration in ug/L

$DA_{event}$  = dermal absorption per swim in  $mg/cm^2$

ND = not detected

NC = not calculated, chemical not detected in ground water

#### *Concentration of COPCs in Air Above Swimming Pool*

The air concentration of COPCs above the pool was calculated to evaluate swimmer inhalation of VOCs over the swimming season. Given the assumed half-life of 3.5 days for VOC volatilization from pool water, the average emission rate of VOCs from a swimming pool containing 1 mg/L of VOC is calculated as

$$\frac{1 \mu\text{g} / \text{L} \times 64,000 \text{L} \times 0.5}{86,400 \text{ seconds} / \text{day} \times 3.5 \text{ days}} = 0.106 \mu\text{g} / \text{s}$$

To calculate a seasonal average emission rate, the emission rate is multiplied by swimming pool loss factor of 0.122 (calculated above) to give a seasonally adjusted emission rate of 0.0127 ug/s (0.106 ug/s x 0.12).

The box model was used to calculate air concentrations above the swimming pool at receptor height. The seasonally adjusted air concentration is 0.000977 ug/m<sup>3</sup> where

Seasonally adjusted emission rate = 0.0129 ug/s

Receptor height above water = 0.5 m

Side of pool perpendicular to the wind = 6.5 m (square root of pool area)

Wind speed = 4 m/s ([http://ggweather.com/ca\\_climate/wind.htm](http://ggweather.com/ca_climate/wind.htm))

$$\frac{0.0127 \mu\text{g} / \text{s}}{0.5 \text{ m} \times 6.5 \text{ m} \times 4 \text{ m/s}} = 0.000977 \mu\text{g} / \text{m}^3$$

A seasonally adjusted swimming pool volatilization factor (VF<sub>pool</sub>) can be calculated as 0.000977 ug/m<sup>3</sup> per 1 ug/L or 0.000977 L/m<sup>3</sup>. This value is used in calculating risk-based concentrations for the chemicals of potential concern in ground water used for swimming pools.

Calculated air concentrations of the VOCs of potential concern over a swimming pool filled with ground water are presented in the table below.

### Modeled Air Concentrations of VOCs Over a Swimming Pool

Volatile Chemical	(a) Bermuda		(b) Bermuda		(c) Stimel		(d) Stimel	
	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc
Acetone	26.0	2.59E-02	2.2	2.19E-03	12.0	1.19E-02	ND	NC
Chloromethane	ND	NC	0.8	8.16E-04	ND	NC	ND	NC
1,1-Dichloroethane	ND	NC	0.2	1.69E-04	ND	NC	ND	NC
1,2-Dichloroethane	ND	NC	ND	NC	ND	NC	ND	NC
1,1-Dichloroethene	11.0	1.09E-02	1.4	1.39E-03	5.0	4.97E-03	4.9	4.87E-03
cis-1,2-Dichloroethene	24.0	2.39E-02	0.3	2.59E-04	17.0	1.69E-02	37.0	3.68E-02
trans-1,2-Dichloroethene	ND	NC	ND	NC	2.4	2.39E-03	ND	NC
Tetrachloroethene	ND	NC	ND	NC	ND	NC	ND	NC
Trichloroethene	670.0	6.67E-01	29.0	2.89E-02	130.0	1.29E-01	210.0	2.09E-01
Vinyl chloride	ND	NC	ND	NC	1.0	9.95E-04	ND	NC

Volatile Chemical	(e) Stimel		(f) Gragg Lane		(g) Thames Dr		(h) Waterloo	
	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc	GW conc	Modeled air conc
Acetone	ND	NC	3.0	2.98E-03	ND	NC	12.0	1.19E-02
Chloromethane	ND	NC	0.9	8.95E-04	ND	NC	ND	NC
1,1-Dichloroethane	0.3	2.89E-04	ND	NC	1.8	1.79E-03	ND	NC
1,2-Dichloroethane	0.3	2.79E-04	ND	NC	ND	NC	ND	NC
1,1-Dichloroethene	1.7	1.69E-03	ND	NC	9.2	9.15E-03	4.4	4.38E-03
cis-1,2-Dichloroethene	12.0	1.19E-02	ND	NC	13.0	1.29E-02	6.5	6.47E-03
trans-1,2-Dichloroethene	0.5	4.87E-04	ND	NC	ND	NC	ND	NC
Tetrachloroethene	ND	NC	ND	NC	ND	NC	ND	NC
Trichloroethene	8.0	7.96E-03	ND	NC	500.0	4.97E-01	380.0	3.78E-01
Vinyl chloride	1.5	1.49E-03	ND	NC	ND	NC	ND	NC

GW conc = ground water concentration in ug/L

Modeled air conc = modeled air concentration from irrigation activities in ug/m<sup>3</sup>

ND = not detected

NC = not calculated, chemical not detected in ground water

### References

USEPA 2001. Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment Interim) Review Draft for Public Comment. September 2001. EPA/540/R/99/005 PB99-963312

## **APPENDIX D**

### **CALCULATION OF AIR CONCENTRATIONS FOR CHEMICALS VOLATILIZING FROM WALNUT CREEK SURFACE WATER**

This appendix describes the calculation of volatile chemical concentrations in ambient air released from Walnut Creek surface water. The method used is from Thomas, 1990. cis-1,2-Dichloroethylene (cis-1,2-DCE), tetrachloroethylene (PCE), and trichloroethylene (TCE) were detected in Walnut Creek surface water in samples collected in 2002 (ERM, 2004).

According to Thomas (1990), emissions from surface water from a stream or river may be calculated by determining the mass transfer coefficient ( $K_L$ ), estimating the emission rate in milligrams of chemical release per second, and calculating a modeled air concentration in milligrams of chemical per cubic meter of air.

#### Calculation of Mass Transfer Coefficient

According to methods described by Thomas (1990), the mass transfer coefficients may be calculated by calculating the gas ( $k_g$ ) and liquid phase ( $k_l$ ) coefficients and using these values to calculate the overall mass transfer coefficient ( $K_L$ ).

$k_g$  (in cm/hr) is calculated as:

$$k_g = 1137.5 \times (V_{wind} + V_{curr}) \times \sqrt{\frac{18}{M}}$$

and

$k_l$  (in cm/hr) is calculated as:

$$k_l = 23.51 \times \left( \frac{V_{curr}^{0.969}}{Z^{0.673}} \right) \times \sqrt{\frac{32}{M}} \times e^{0.526 \times (V_{wind} - 1.9)}$$

and

$K_L$  (in cm/hr) is calculated as:

$$K_L = \frac{H' \times k_g \times k_l}{(H' \times k_g) + k_l}$$

Where:

$V_{\text{wind}}$  = windspeed in m/s; a value of 4 m/s was assumed for the Walnut Creek area

$V_{\text{curr}}$  = stream flow velocity in m/s; a value of 1 m/s was assumed for Walnut Creek

M = molecular weight of the chemical of interest

Z = depth of the stream in meters; the depth of Walnut Creek was assumed to be 0.61 m

H' = Henry's Law constant, unitless; values for cis-1,2-DCE, PCE, and TCE are 0.167, 0.754, 0.422, respectively.

Emissions of VOCs from Walnut Creek surface water may be calculated by the equation

$$E = K_L \times \text{hr}/3600\text{s} \times \text{CW} \times 1 \text{ L}/1000 \text{ cm}^3 \times A$$

Where:

E = emission rate in ug/s

$K_L$  = the calculated chemical-specific mass transfer coefficient in cm/hr

CW = concentration of VOC in surface water in ug/L

A = area in  $\text{cm}^2$ ; the area of the stream assumed to continuously emit VOCs is assumed to be 30 feet wide (914 cm) by the length of a 0.5 acre lot (about 148 feet or 4500 cm) =  $4.11 \times 10^6 \text{ cm}^2$

Air concentrations of the VOCs near the stream (CA) were calculated using a simple box model as

$$CA = \frac{E}{LS \times V \times MH}$$

Where:

CA = concentration of VOC in air in  $\text{ug}/\text{m}^3$

E = emission rate in ug/s

LS = length of the side perpendicular to the wind; this value is assumed to be the length of a side of a 0.5 acre lot (45 meters)

MH = mixing height; assumed to be 2 m

Calculated  $k_g$ ,  $k_l$ ,  $K_L$ , E, and CA values are presented in the attached table.

#### Reference

Thomas, R.G. 1990. Chapter 15. Volatilization from Water. In: *Handbook of Chemical Property Estimation Methods*. Ed: W. J. Lyman, W. F. Reehl, and D. H. Rosenblatt. American Chemical Society. Washington, D.C.

**Calculation of VOC Concentrations in Air Emitted from Surface Water  
Inputs and Calculated Constants and Concentrations**

Volatile Chemicals	Surface Water Concentration ug/L	Gas phase Transfer Coefficient ( $k_g$ ) cm/hr	Liquid phase transfer coefficient ( $k_l$ ) cm/hr	Overall Mass Transfer Coefficient ( $K_L$ ) cm/hr	Emission rate (E) ug/s	Calculated Air Concentration (CA) ug/m <sup>3</sup>
cis-1,2-Dichloroethylene	1.4	2450	56.84	49.91	79.82	0.222
Tetrachloroethylene	2.6	1873	43.45	42.15	125.2	0.348
Trichloroethylene	3.3	2108	48.91	46.36	174.8	0.486

**APPENDIX E**

**ESTIMATION OF LEAD EXPOSURE**



Unlike other chemicals for which human exposure is calculated in terms of chemical intake (intake in milligrams of chemical per kilogram of body weight per day, mg/kg/day), exposure and risks associated with exposure to lead are based on an estimated blood lead concentration. Due to the existence of a growing database relating blood lead concentration (typically expressed in terms of micrograms of lead per deciliter of blood, µg/dL) and human toxicity, blood lead concentration is the most direct means by which the toxic effects of lead in humans can be assessed.

The State of California DTSC, United States Environmental Protection Agency (EPA), and others have developed lead exposure models for evaluating blood lead concentrations associated with intake of lead from food, water, air, and soil. An updated version of California DTSC lead exposure model (Leadsread 7) was used in this HRA to predict blood lead increases for the future on-site resident and construction worker. The model also includes lead exposure from background sources including air, food, and water.

The DTSC uses a 10 µg/dL blood lead concentration as its “concentration of concern” in both children and adults. This level is consistent with USEPA’s guidance regarding lead exposure in children. However, this level is lower than California Occupational and Health Administration’s (Cal OSHA) guidance with regard to construction workers. CCR (California Code of Regulations) Title 8 § 1532.1 requires that a construction worker be removed for medical reasons if any blood lead test on the worker yields a blood lead concentration that is at or above 50 µg/dL. A blood lead concentration of 40 µg/dL to 49 µg/dL triggers several employee notification requirements. Appendix A to §1532.1 indicates that maintaining a blood lead concentration below 30 µg/dL is a “health protection goal”.

Thus, while the regulation does not establish a medical removal for a blood lead concentration level less than 40 µg/dL, it does recommend that worker blood lead levels be lower than 30 µg/dL. Thus, Cal OSHA regulations allow for higher levels of worker exposure than the current DTSC point of departure of 10 µg/dL.

The site-specific exposure inputs used in the DTSC lead exposure model are presented in table below. All other exposure inputs used in the DTSC lead exposure model were DTSC default residential or occupational values.

Calculated blood lead concentrations for the future on-site resident, future on-site worker, and future construction worker are presented in the attached tables. Tables 1 and 2 present blood lead calculation results for the on-site commercial/industrial worker and construction worker exposed to lead in soil in the 0 to 10 feet bgs depth range. The calculated blood lead

concentrations were below 10 µg/dL for both workers even at the 99<sup>th</sup> percentile of calculated blood lead concentrations.

**\*Site-Specific Exposure Parameters for Lead Exposure Model**

Parameter	Input	Source/Comment
<b>Lead in Soil/Dust</b>	0-10' bgs soils = 104 ug/g	Calculated 95% upper confidence limit on the arithmetic mean concentrations of soil lead
<b>Lead in water</b>	1 µg/L	The 90 <sup>th</sup> percentile at the tap drinking water lead concentrations was "ND" Annual Water Quality Report 2003 Contra Costa County Water District. The 1 ug/L value is one-half the State of California Public Health Goal for drinking water.
<b>Respirable dust</b>	<u>Construction worker</u> 700 µg/m <sup>3</sup>	Construction worker RWQCB default
<b>Soil Ingestion</b>	<u>Construction worker</u> 165 mg	Soil ingestion was assumed to be one-half of default upper bound soil ingestion rate for construction workers (330 mg/day) in keeping with the use of central tendency estimates of soil exposure. For the purpose of blood lead exposure calculations, the DTSC sets soil ingestion rates for the child and adult resident at one-half of the USEPA default upper bound value.

\*Unless specified in this table, default Leadsread exposure parameter used

Table 1  
On-Site Commercial/Industrial Worker Soil Exposure-0 to 10 feet bgs Soil  
**LEAD RISK ASSESSMENT SPREADSHEET**  
CALIFORNIA DEPARTMENT OF TOXIC SUBSTANCES CONTROL

USER'S GUIDE to version 7

INPUT	
MEDIUM	LEVEL
Lead in Air (ug/m <sup>3</sup> )	0.028
Lead in Soil/Dust (ug/g)	104.0
Lead in Water (ug/l)	1
% Home-grown Produce	7%
(ug/m <sup>3</sup> )	1.5

OUTPUT							
Percentile Estimate of Blood Pb (ug/dl)						PRG-99	PRG-95
50th	90th	95th	98th	99th	(ug/g)	(ug/g)	
BLOOD Pb, OCCUPATION,	0.4	0.7	0.8	1.0	1.2	4692	6681

EXPOSURE PARAMETERS			
	units	adults	children
Days per week	days/wk	7	
Days per week, occupational		5	
Geometric Standard Deviation		1.6	
Blood lead level of concern (ug/dl)		10	
Skin area, residential	cm <sup>2</sup>	5700	2900
Skin area occupational	cm <sup>2</sup>	2900	
Soil adherence	ug/cm <sup>2</sup>	70	200
Dermal uptake constant (ug/dl)/(ug/cm <sup>2</sup> )		0.0001	
Soil ingestion	mg/day	50	100
Soil ingestion, pica	mg/day		200
Ingestion constant (ug/dl)/(ug/cm <sup>2</sup> )		0.04	0.16
Bioavailability	unitless	0.44	
Breathing rate	m <sup>3</sup> /day	20	6.8
Inhalation constant (ug/dl)/(ug/cm <sup>3</sup> )		0.08	0.19
Water ingestion	l/day	1.4	0.4
Food ingestion	kg/day	1.9	1.1
Lead in market basket	ug/kg	3.1	
Lead in home-grown produce	ug/kg	46.8	

Click here for REFERENCES

PATHWAYS						
ADULTS	Residential			Occupational		
	Pathway contribution			Pathway contribution		
	PEF	ug/dl	percent	PEF	ug/dl	percent
Soil Contact	3.8E-5	0.00	#DIV/0!	1.4E-5	0.00	0%
Soil Ingestion	8.8E-4	0.09	#DIV/0!	6.3E-4	0.07	17%
Inhalation, bkgrnd		0.05	#DIV/0!		0.03	8%
Inhalation	2.5E-6	0.00	#DIV/0!	1.8E-6	0.00	0%
Water Ingestion		0.06	#DIV/0!		0.06	14%
Food Ingestion, bkgrnd		0.22	#DIV/0!		0.23	60%
Food Ingestion	2.4E-3	0.25	#DIV/0!			0%
CHILDREN	typical			with pica		
	Pathway contribution			Pathway contribution		
	PEF	ug/dl	percent	PEF	ug/dl	percent
Soil Contact	5.6E-5	0.01	#DIV/0!		0.01	#DIV/0!
Soil Ingestion	7.0E-3	0.73	#DIV/0!	1.4E-2	1.46	#DIV/0!
Inhalation	2.0E-6	0.00	#DIV/0!		0.00	#DIV/0!
Inhalation, bkgrnd		0.04	#DIV/0!		0.04	#DIV/0!
Water Ingestion		0.06	#DIV/0!		0.06	#DIV/0!
Food Ingestion, bkgrnd		0.50	#DIV/0!		0.50	#DIV/0!
Food Ingestion	5.5E-3	0.58	#DIV/0!		0.58	#DIV/0!

Table 2  
On-Site Construction Worker Soil Exposure-0 to 10 feet bgs Soil  
**LEAD RISK ASSESSMENT SPREADSHEET**  
CALIFORNIA DEPARTMENT OF TOXIC SUBSTANCES CONTROL

USER'S GUIDE to version 7

INPUT	
MEDIUM	LEVEL
Lead in Air (ug/m <sup>3</sup> )	0.028
Lead in Soil/Dust (ug/g)	104.0
Lead in Water (ug/l)	1
% Home-grown Produce	7%
(ug/m <sup>3</sup> )	700

OUTPUT							
Percentile Estimate of Blood Pb (ug/dl)					PRG-99	PRG-95	
50th	90th	95th	98th	99th	(ug/g)	(ug/g)	
BLOOD Pb, OCCUPATION,	0.6	1.1	1.4	1.6	1.9	1039	1480

EXPOSURE PARAMETERS			
	units	adults	children
Days per week	days/wk	7	
Days per week, occupational		5	
Geometric Standard Deviation		1.6	
Blood lead level of concern (ug/dl)		10	
Skin area, residential	cm <sup>2</sup>	5700	2900
Skin area occupational	cm <sup>2</sup>	2900	
Soil adherence	ug/cm <sup>2</sup>	70	200
Dermal uptake constant (ug/dl)/(ug/cm <sup>2</sup> )		0.0001	
Soil ingestion	mg/day	165	100
Soil ingestion, pica	mg/day		200
Ingestion constant (ug/dl)/(ug/cm <sup>2</sup> )		0.04	0.16
Bioavailability	unitless	0.44	
Breathing rate	m <sup>3</sup> /day	20	6.8
Inhalation constant (ug/dl)/(ug/cm <sup>3</sup> )		0.08	0.19
Water ingestion	l/day	1.4	0.4
Food ingestion	kg/day	1.9	1.1
Lead in market basket	ug/kg	3.1	
Lead in home-grown produce	ug/kg	46.8	

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PATHWAYS						
ADULTS	Residential			Occupational		
	Pathway contribution			Pathway contribution		
	PEF	ug/dl	percent	PEF	ug/dl	percent
Soil Contact	3.8E-5	0.00	#DIV/0!	1.4E-5	0.00	0%
Soil Ingestion	2.9E-3	0.30	#DIV/0!	2.1E-3	0.22	35%
Inhalation, bkgrnd		0.05	#DIV/0!		0.03	5%
Inhalation	1.1E-3	0.12	#DIV/0!	8.2E-4	0.09	14%
Water Ingestion		0.06	#DIV/0!		0.06	9%
Food Ingestion, bkgrnd		0.22	#DIV/0!		0.23	37%
Food Ingestion	2.4E-3	0.25	#DIV/0!			0%
CHILDREN	typical			with pica		
	Pathway contribution			Pathway contribution		
	PEF	ug/dl	percent	PEF	ug/dl	percent
Soil Contact	5.6E-5	0.01	#DIV/0!		0.01	#DIV/0!
Soil Ingestion	7.0E-3	0.73	#DIV/0!	1.4E-2	1.46	#DIV/0!
Inhalation	9.1E-4	0.10	#DIV/0!		0.10	#DIV/0!
Inhalation, bkgrnd		0.04	#DIV/0!		0.04	#DIV/0!
Water Ingestion		0.06	#DIV/0!		0.06	#DIV/0!
Food Ingestion, bkgrnd		0.50	#DIV/0!		0.50	#DIV/0!
Food Ingestion	5.5E-3	0.58	#DIV/0!		0.58	#DIV/0!